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The Next Annual Meeting of the Massachusetts Medical Society Will Be Held in Springfield, June 8, 9, 10, 1936, at the Hotel Kimball and the Springfield Auditorium

The New England JOURNAL of MEDICINE

FORMERLY THE BOSTON MEDICAL AND SURGICAL JOURNAL

Official Organ of The Massachusetts Medical Society
The New Hampshire Medical Society—The Vermont State Medical Society
The New England Surgical Society—The Boston Surgical Society, Inc.
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OWNED AND PUBLISHED BY THE MASSACHUSETTS MEDICAL SOCIETY

Volume 214 Number 16 | Domestic $6 per Annum, 25c per Copy Canada $7.04, Foreign $8.52 | APRIL 16, 1936 | Published Weekly in Boston at 8 Fenway

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The New England
Journal of Medicine

VOLUME 214 APRIL 16, 1936 NUMBER 16

PROGRESS IN THE RECOGNITION OF CONGENITAL HEART DISEASE*

BY SYLVESTER MC GINN, M.D.,† AND PAUL D. WHITE, M.D.†

OUR knowledge of cardiovascular disease has increased in all directions during the past decade. The study of congenital heart disease has shared in this progress and has become of great importance as well as of great interest. Although abnormalities of the heart present at birth have been known to exist for centuries, contributions on the subject have largely been confined to postmortem observations. It is in recent years only, during which time we have been aided by the fluoroscope and electrocardiograph, that we have been able to make the diagnosis clinically with any degree of confidence. The time has now arrived when it is not sufficient to make the broad diagnosis of congenital heart disease without having definite structural defects in mind.

We have recently reviewed 7500 autopsy records at the Massachusetts General Hospital to determine the frequency with which congenital heart disease has been occurring there since 1895. In addition, we have more closely analyzed the cases coming to autopsy in the last fifteen years to judge the accuracy of our relatively recent clinical diagnoses of congenital heart disease.

A. Incidence of Congenital Heart Disease in the Postmortem Examinations of the Massachusetts General Hospital.

In 7500 postmortem examinations performed in the last forty years, congenital heart disease was found sixty-seven times, representing an incidence of 0.9 per cent. Only definite cases of congenital defects were included, thereby eliminating a large group of cases with foramina ovales functionally closed but patent to the passage of a probe. About one-third (twenty-one) of the sixty-seven patients were infants under one year of age.

Thirty-four hundred of the 7500 autopsies were made in the last fifteen years, and the clinical records of the forty-one cases having congenital lesions of the heart present among these autopsies were subjected to further study. The incidence of congenital heart disease in the latter group was 1.2 per cent, whereas only half as many, or 0.6 per cent, were observed in the preceding 4100 cases. This suggests that more congenital defects can be found with more careful examination of the heart.

Lesions permitting a flow of blood between the arterial and venous systems were found in twenty hearts (about half). The most common congenital defect of all in the 3400 autopsies of the last fifteen years was patency of the ductus arteriosus which occurred alone in four cases and in combination in five others. An interauricular septal defect occurred alone in one case and in combination in six others. These two lesions were associated five times. One of the hearts with a patent ductus arteriosus also had coarctation of the aorta. One of the cases with imperfect closure of the auricular septum had mitral stenosis in addition, a combination known as Lutembacher's disease.

Four hearts had interventricular septal defects, and two others were complicated by pulmonary stenosis, dextroposition of the aorta, and enlarged right ventricle, comprising the tetralogy of Fallot. In one heart there were defects in both the auricular and ventricular septa. One three chambered-heart was found, a cor biatriatum triloculare, and there was one two-chambered heart, a cor biloculare. Three cases of congenital idiopathic hypertrophy were described. Abnormalities of the coronary vessels were noted in five cases.

Abnormalities in the heart valves accounted for thirteen of the forty-one cases. Bicuspid aortic valves were noted in seven hearts, a bicuspid pulmonary valve in one, pulmonary valves with four cusps in two, and pulmonary stenosis, single mitral leaflet, and variation in the size of the aortic cusps in one case each.

In contrast to this group of forty-one cases observed during the years 1921 to 1935 inclusive, congenital defects were discovered in twenty-six patients between 1896 and 1920, during which time 4100 postmortem examinations were performed. In eight hearts uncomplicated defects in the interauricular septum were present. Uncomplicated patent ductus arteriosus and interventricular septal defect were found once each. Pulmonary stenosis was associated with an interauricular septal defect in two cases, and with an interventricular defect once. In eight cases there were patencies in both the interauricular septum and the duc-

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tus arteriosus. In one heart there was an interauricular septal defect associated with mitral stenosis. A bicuspid aortic valve was found three times, and in one case it was noted that the pulmonary valve had four cusps.

The apparent discrepancy in the incidence of congenital lesions in these two groups seems to rest upon the observations of the coronary vessels and of the heart valves. The number of defects allowing admixture of blood were about equal in the two groups. The coronary vessels have been inspected much more carefully by the pathologists in recent years. Evidence of this is illustrated by the five cases of abnormal coronary arteries observed in the last fifteen years, in contrast to the failure to note any prior to that time. The relative incidence of congenital defects of the valves in the earlier and later groups were four and thirteen respectively.

B. Accuracy of the Clinical Diagnosis of Congenital Heart Disease.

The clinical records of the forty-one cases examined postmortem in the last fifteen years were analyzed to estimate the accuracy of our diagnoses. A correct diagnosis was made seven times and included four adults and three infants under one year of age. The cases in this group consisted of three with idopathic congenital hypertrophy, two with interventricular septal defect (one of these had also a patent foramen ovale), one with the tetralogy of Fallot, and one with pulmonary stenosis.

In five other cases, including one infant, the correct diagnosis was suspected and so entered on the record. This group comprised three cases with an interventricular septal defect, one with an interauricular septal defect, and one with a patent ductus arteriosus. These twelve cases in which a correct diagnosis was made or very definitely suspected represent 29 per cent of the congenital lesions coming to autopsy in the last fifteen years and include four of the eleven infants, or 36 per cent of those under one year of age. The correct diagnosis of congenital cardiovascular defects is, however, very difficult in infants under six months of age, as has been indicated by studies at the Children's Hospital in Boston.

In three cases a diagnosis of congenital heart disease was made, but the anatomical defect was not noted. One of these cases had a cor biloculare. The two others showed congenital hypertrophy diagnosed as idiopathic, but one had an abnormal coronary supply and the other had a patent ductus arteriosus.

One case was diagnosed wrongly as to the congenital structural defect in the heart, viz., a case of tetralogy of Fallot which was believed to have a patent ductus arteriosus.

In the remaining twenty-five cases, including four infants, the diagnosis of congenital heart disease was entirely unsuspected. Thirteen of these hearts had defects in the structure of one of the valves, seven of them having bicuspid aortic valves. There were four cases with abnormal variations in the coronary vessels. There were two cases of patent ductus arteriosus alone and five in which it was associated with an interauricular septal defect. Finally, a case of cor triloculare was thought to have rheumatic heart disease.

Of the twenty-three cases of idiopathic hypertrophy or of congenital defects allowing admixture of blood, the correct diagnosis of specific structural defects was made or suggested in eleven (48 per cent).

The correct diagnosis was not made or suggested in any of the twenty-six cases of congenital heart disease found in the first 4100 postmortem examinations. In two cases the presence of congenital heart disease was suspected, but the specific lesion was not suggested. One of these cases had an interauricular septal defect associated with mitral stenosis, and the other case was one of pulmonary stenosis with septal defects.

A third group of cases was analyzed for comparative purposes and included all of the cases of congenital heart disease proved at autopsy that had been previously examined by members of the staff of the cardiac clinic in or outside the hospital. A majority of these was included in the 7500 autopsies reviewed. A correct diagnosis was made or suggested in eleven of the nineteen cases (58 per cent): it was made in two cases of the tetralogy of Fallot, two cases with interventricular septal defects, one case of patent ductus arteriosus, one case of idiopathic hypertrophy, and one case of pulmonary stenosis, and was suggested in three cases of interventricular septal defect and in one case of interauricular septal defect. The correct structural diagnosis was unsuspected in six cases (although in one of these a finding of congenital heart disease was made): three with bicuspid aortic valves, one with a pulmonary bicuspid valve, one with a single mitral leaflet, and one with an anomalous coronary vessel. If these six cases, which gave no characteristic clinical signs, are eliminated, the correct diagnosis was made or suggested in 85 per cent of this small group of cases.

DISCUSSION

In reviewing our series of cases with the aim of improving our diagnostic ability, we are confronted with a large group of lesions, namely, those of defective valve structure and abnormal coronary vessels, which at the present time provide no evidence to suggest their presence. There is some importance in recognizing abnormalities of the valves, not that they always interfere seriously with the cardiac function but because they so frequently are the

foci for subacute bacterial endocarditis. The bicuspid aortic valve is the most common and most important of the group, and so far as we know has no clinical manifestation. Other valvular defects, such as pulmonary valves with four cusps, bicuspid pulmonary valves, and mitral valves with a single leaflet, give no clinical evidence of which we are aware. As yet we are unable to recognize, with one possible exception to be noted below, hearts supplied by a single or anomalous coronary vessel. Further observation of this rather large number of cases with silent congenital lesions is necessary before we can improve much further our accuracy in diagnosing congenital heart disease.

Within recent years congenital cardiac lesions have provided sufficient evidence of some defects and suggestive evidence of other defects to permit us to make the diagnosis with certainty or at least to suspect that it is present. More experience will eventually allow us to be more confident. The x-ray and electrocardiograph have been very helpful in obtaining further clinical evidence.

We believe that it will prove helpful to present herewith the diagnostic clues to some of the individual and combined defects of congenital heart disease with which we have recently become more familiar, as well as to the very few defects that have been clinically recognized for many years. We shall begin with the latter.

1. *Patent ductus arteriosus.* The ductus arteriosus which connects the pulmonary artery and aorta in the fetus normally atrophies two or three months after birth and is later represented by a fibrous ligament. The failure of this vessel to close, results in a permanent shunt between the greater and lesser circulations. Such a shunt is often associated with other anomalies and not infrequently has a compensatory function.

The most characteristic clinical sign of this condition is a typical murmur. It is heard best, and sometimes only, in the second left interspace, and is a harsh humming murmur with a sound similar to a mill wheel or the machine-like purr of a large motor. It is continuous with accentuation during systole, and may be accompanied by a thrill.

Cyanosis and clubbing of the fingers are usually absent. Cardiac enlargement, especially involving the right ventricle, may be present and consequently may produce right axis deviation in the electrocardiogram. X-ray examination is usually characteristic and sometimes pathognomonic with a variable degree of prominence in the region of the pulmonary conus: fluoroscopy may show a pulsation of the lung hilus shadows.

2. *Interventricular septal defects.* A defect in the interventricular septum (Roger's disease, 1879) is one of the most common congenital lesions to be recognized clinically. The defect is usually a patency in the interventricular septum, one or two centimeters in diameter, situated just below the aortic valve and allowing a free passage of blood between the two ventricles. Other malformations of the heart frequently complicate this condition.

Cyanosis is a rare finding in the uncomplicated cases. The left ventricle is stronger than the right, so that the tendency is for the oxygenated arterial blood to be shunted through the opening to the venous side of the circulation. This condition persists until the left ventricle fails.

There are several findings which may lead one to make the correct clinical diagnosis. The most important evidence is the presence of a harsh systolic murmur heard best at the third left interspace and more or less confined to that immediate area but frequently heard in the back between the scapulae. There is usually a thrill accompanying the murmur. Any intense murmur heard in infancy justifies the assumption that it is due to a lesion present since birth, and defects of the interventricular septum are among the most common congenital lesions detected clinically. This information may have to be obtained from the obstetrician if the patient is beyond infancy. X-ray examination fails to show gross enlargement despite the loud murmur. There is an unusual blunting of the apex, probably due to the changes in the right ventricle, but it is not a pathognomonic finding.

3. The term *idiopathic congenital hypertrophy of the heart* refers to enlarged and dilated hearts observed at birth or early infancy and for which no cause can be discovered.

4. *The tetralogy of Fallot* is a fairly common congenital lesion that we can now recognize. It consists of (a) stenosis or narrowing of the opening of the pulmonary artery at the valve cusps or of the infundibulum just below it (comparable to the fibrous ridge found in subaortic stenosis), (b) an interventricular septal defect, (c) dextroposition of the aorta, which means that the aortic opening is moved farther to the right than is normal and overlies the septal opening, and (d) hypertrophy of the right ventricle.

The course of the blood flow in the heart is as follows: the venae cavae empty into the right auricle from which the blood flows into the right ventricle; at this point the flow of blood, obstructed by the narrow pulmonary orifice, only in part enters the pulmonary artery, the balance going directly into the aortic orifice which rides over the interventricular septal defect and so receives blood from both ventricles. The right ventricle hypertrophies to accommodate itself to this increased work and the aorta is larger than normal. Cyanosis results from

the large quantity of unaerated blood being shunted into the arterial circulation. The cyanosis is usually intense but occasionally it does not become apparent, except in paroxysms, until later life. The lungs receive less blood than normally and pass it on to the left auricle and left ventricle which may be somewhat small. Thence the oxygenated blood enters the aorta along with the unoxygenated blood from the right ventricle.

There are five findings, which when they occur in association permit the diagnosis of the tetralogy of Fallot with a high degree of certainty. First, there is usually cyanosis of lips, cheeks, ears, fingers, and toes; secondly, there is clubbing of the fingers and toes; thirdly, a loud systolic murmur is heard best in the pulmonary valve area and in the third left interspace (at times accompanied by a systolic thrill); fourthly, there is marked right axis deviation by electrocardiogram in as extreme a degree as we ever see; and fifthly, x-ray examination shows the heart to be sabot-shaped due to enlargement of the right ventricle without enlargement of the pulmonary artery. (The great vessels will be prominent on the right due to the dextroposition of the aorta but not to the left because of the small amount of blood passing through the hypoplastic pulmonary artery.)

The prognosis may be good, for we have seen instances of a noted musician who died in his sixtieth year, the diagnosis proved by necropsy, and of a woman now sixty-two years old who works as a librarian without complaints other than that of cyanosis. The average age at death in eighty-five cases was twelve and one-half years (Abbott).

There are five other congenital lesions found in our postmortem series that have clinical evidence sufficient to allow one to suspect the diagnosis. As time will give us further opportunity to study similar cases, we may make these diagnoses with confidence comparable to that which we feel in the more common lesions.

5. *Coarctation of the aorta*, or narrowing of the lumen of the aorta, is a congenital anomaly with which we have become familiar in recent years and which we can now diagnose clinically with ease if it is at all pronounced. The narrowing of the aorta most frequently occurs at the point where the ductus arteriosus joins it. The ductus normally disappears leaving a fibrous ligament in its place. It is believed that this fibrous or atrophic change involves the walls of the aorta and narrows its lumen. This materially interferes with the circulation beyond the point of narrowing, diminishing the volume of blood going to the lower extremities and necessitating a collateral circulation. The internal mammary and intercostal arteries become much larger than normal and by a circuitous route supply blood to the lower parts of the body and thus comprise the chief factors in the collateral circulation.

There are five points that make the clinical diagnosis possible. (1) Hypertension occurring in children and young adults should make one suspicious of aortic coarctation and should direct the search along that line. (2) The blood pressure is higher in the arms than it is in the lower extremities and the dorsalis pedis, tibial, and popliteal pulsations may be absent. Normally the blood pressure in the legs is considerably higher than in the arms. (3) The blood vessels of the trunk, especially the intercostal and mammary arteries, can be seen and palpated as they pulsate vigorously. (4) A harsh systolic murmur can often be heard at the base of the heart and in the back. (5) X-ray examination may show evidence of left ventricular hypertrophy and especially of notches in the ribs made by the dilated intercostal arteries. The x-ray finding of rib notching is pathognomonic.

The prognosis in these cases is varied, the oldest subject on record being ninety-two years of age. Ultimately the patients may die of cardiac failure, rupture of the aorta, or subacute bacterial endocarditis to which they are especially susceptible.

6. *The persistence of a right aortic arch* is another congenital anomaly with which we have become familiar in the past few years. There are several variations of this condition, but in brief the aorta starts from the left ventricle in its usual position, ascends to the right of the sternum, but at the arch it courses to the right rather than to the left and then finally swings behind the esophagus and trachea to the left of the spine where it descends through the thorax into the abdomen. Both the esophagus and trachea are subjected to the pressure of the transverse aortic arch behind and to the remnant of the ductus arteriosus (the ligamentum arteriosum) on the left side. The ligament runs from the aorta to the pulmonary artery and completes an encircling and constricting collar around the trachea and esophagus.

The presence of symptoms depends on the degree of constriction. Although most patients have no complaints and the anomaly is picked up in a routine examination, occasionally it causes dysphagia, esophageal ulcerations, respiratory distress, and even asphyxia by compression. We have recently observed a patient thought to have carcinoma of the esophagus and another who was considered to be a neurasthenic individual with globus hystericus, both of whom had this abnormality. The diagnosis can be easily made by x-ray examination with barium in the esophagus. Three findings will be observed by x-ray examination; in the anteroposterior view the ascending aorta and the aortic knob will be found to the right of the sternum; the barium filled esophagus will be displaced to the left; and in the oblique view the esopha-

gus will be pushed forward. A surgical procedure has been suggested to section the ligamentum arteriosum but we do not know of its having been done as yet.

7. *Coronary artery anomalies.* In one of our autopsy cases we encountered a large heart in an infant dying at the age of three months. The clinical diagnosis was idiopathic congenital hypertrophy which proved to be incorrect when, at autopsy, a single coronary artery was found arising from the pulmonary artery. The myocardium was in this case being supplied with venous rather than oxygenated blood. This nutritional disturbance and relative anoxemia of the heart muscle was reflected clinically by attacks of distress on effort like nursing (probably angina pectoris) and in the electrocardiogram by inverted T waves of the coronary type and analogous to those found when there is anoxemia of the heart muscle as the result of coronary occlusion and myocardial infarction. In the future when confronted with the findings of congenital hypertrophy and inversion of the T wave we shall consider this diagnosis as probable.

8. *A cor biatriatum triloculare*, or a three chambered heart with two auricles and a single ventricle, was found once in our autopsy series. It was wrongly diagnosed as rheumatic heart disease with pericardial effusion. In the future a heart with a "water bottle" shape similar to the x-ray picture of this case, associated with the conduction disturbance of intraventricular block in the electrocardiogram, will make us suspicious of a cor triloculare.

9. Defects in the septum separating the two auricles, or *interauricular septal defects*, are among the commonest of congenital cardiac lesions found at postmortem examinations. The foramen ovale, so important in the fetal circulation, does not become entirely closed until the eighth month after birth. It has been estimated that in 25 per cent of the hearts seen at postmortem examination the foramen ovale is open sufficiently to allow passage of a probe. However, this type is unimportant and we refer to an actual aperture of a centimeter or more when we speak of an interauricular septal defect.

(a) Although interauricular septal defects are not uncommon at necropsy, the diagnosis is rarely made clinically, largely due to the fact that they are symptomless unless the lesion is of extreme degree. There are four findings to suggest this anomaly. First, late cyanosis (cyanose tardive), usually intense, occurs with severe illness or at approaching death and may be due to an interauricular septal defect. Whatever exchange of blood ordinarily takes place is from the left to the right auricle. When the right ventricle fails, a reversal of flow follows with a consequent cyanosis, due to increased right auricular pressure. Secondly, the x-ray of the heart shows a large pulmonary artery, enlarged lung hilus shadows, and an enlarged right ventricle. The pulmonary artery or one of its main branches may become so dilated that it is mistaken for a pulmonary aneurysm. Thirdly, the electrocardiogram shows right axis deviation. Fourthly, there may be "paradoxical embolism", a condition characterized by the passage of emboli from the systemic veins or right auricle into the systemic arterial circuit through an auricular septal defect. There are no murmurs to be heard with the stethoscope.

By the time there is sufficient evidence to make a clinical diagnosis, the heart has undergone extensive changes and the prognosis is poor.

(b) *Interauricular septal defects in association with mitral stenosis* are rarely found, there being only twenty-four reported in the literature, two of which were noted at the Massachusetts General Hospital. It is worth while to discuss the condition because it illustrates so well alterations in blood flow. Because of stenosis of the mitral valve the pressure of blood in the left auricle rises. A considerable portion of blood passes through the interauricular septal defect rather than through the slit-like mitral valve. This blood passes from the right auricle into the right ventricle, and thence to the lungs for a second time. As a consequence of doing double duty, the right ventricle hypertrophies and the pulmonary artery dilates. The aorta, on the other hand, remains small and aplastic because it is receiving but a small amount of blood.

X-ray study is the most important aid in making the diagnosis clinically and it shows the changes mentioned above, namely, a large heart due to an hypertrophied right ventricle, a dilated pulmonary conus, an enlarged right auricle, and a small aplastic aorta. The hilus shadows are also enlarged, to such an extent that they have been mistaken for the mediastinal glands of Hodgkin's disease and unsuccessfully given x-ray therapy. The electrocardiogram shows right axis deviation.

It is evident that cyanosis is at first unlikely in this condition, since the blood is actually being aerated in the lungs twice. Eventually, however, the right ventricle may fail and blood then backs up into the right auricle increasing the pressure within that chamber. A reversal of flow then takes place with venous blood pouring through the interauricular septal defect into the left heart chambers and systemic arteries to cause cyanosis. Finally the venous pressure is increased, as is indicated by dilated neck veins and an engorged, tender, pulsating liver. An enlarged heart is easily detected on physical examination and a mitral diastolic murmur can be heard.

SUMMARY

A review has been made of 7500 postmortem examinations and their clinical records at the Massachusetts General Hospital to determine the incidence of congenital heart disease and the accuracy of diagnosis before death. Congenital defects were found in sixty-seven hearts, or 0.9 per cent, of which twenty-one were infants under one year of age. The correct diagnosis was not made or suggested in any of the twenty-six cases found in the first 4100 (seen before fifteen years ago). In the last fifteen years forty-one hearts with congenital lesions were found in 3400 autopsies and the correct diagnosis was made or suggested in 29 per cent, and for the group of infants under one year of age in 36 per cent. The members of the cardiac clinic staff examined nineteen cases of congenital heart disease that subsequently came to autopsy and made or suggested the correct diagnosis in 58 per cent. Abnormalities of the heart valves and anomalous coronary arteries comprised a large portion of the cases and undoubtedly prevented a higher percentage of accuracy in clinical diagnoses because of the failure of these lesions to give clinical signs. If these lesions were omitted, the correct diagnosis was made or suggested in 48 per cent and 85 per cent respectively of the two series of cases analyzed.

We have presented clues to the diagnosis of the more important congenital cardiovascular defects: (1) patent ductus arteriosus, (2) interventricular septal defects, (3) idiopathic congenital hypertrophy of the heart, (4) the tetralogy of Fallot, (5) coarctation of the aorta, (6) the persistence of a right aortic arch, (7) coronary artery anomalies, (8) cor biatriatum triloculare, and (9) interauricular septal defects, with and without mitral stenosis.

CLASSIFICATION OF CONGENITAL HEART LESIONS NOTED AS FOUND IN 7500 AUTOPSIES AT THE MASSACHUSETTS GENERAL HOSPITAL

	Autopsies 1 to 4100	Autopsies 4100 to 7500	Total
Defects due to incomplete closure of septa and ductus arteriosus			
Interauricular defects alone	8*	1	9
Interventricular defects alone	1	4	5
Patent ductus arteriosus alone	1	4	5
Patent ductus arteriosus and interauricular septal defect	8	5	13
Interventricular septal defect and interauricular septal defect	0	1	1
Pulmonary stenosis and interauricular septal defect	2	0	2
Pulmonary stenosis, interventricular septal defect, dextroposition of aorta, and right ventricular hypertrophy (tetralogy of Fallot)	1	2	3
Mitral stenosis and interauricular septal defect	1	1	2
Cor biatriatum triloculare	0	1	1
Cor biloculare	0	1	1
Idiopathic congenital hypertrophy	0	3	3
Abnormal coronary arteries			
Anomalous	0	2	2
Solitary	0	2	2
Accessory	0	1	1
Valvular defects			
Pulmonary stenosis (uncomplicated)	0	1	1
Pulmonary valve with four cusps	1	2	3
Bicuspid aortic valve	3	7	10
Bicuspid pulmonary valve	0	1	1
Variation in size of aortic cusps	0	1	1
Single mitral leaflet	0	1	1
	26	41	67

*This figure includes cases with defects in the foramen ovale.

THE INCREASE IN CORONARY DISEASE AND ITS CAUSE

BY FRANCIS P. DENNY, M.D.*

THERE is frequent reference in the literature to the increase in mortality from disease of the coronary arteries. It is probably the impression of most physicians who have been in practice for the past twenty years that more of their patients are now dying of coronary disease than formerly.

Statistical evidence of this increase except during the past five years is very meagre, because the International Classification of Deaths, until its 1930 revision, had no subdivision for diseases of the coronary artery and this important group was included under "Other diseases of the heart" (No. 90 of the 1920 Classification) which included every form of heart disease except acute endocarditis, acute myocarditis and angina pectoris. This was an impossible grouping from the etiological standpoint. It is therefore only since 1930 that it has been possible to determine how many deaths from disease of the coronary artery there have been in the registration area of the United States, in the states, or in the various municipalities.

With the object of making available some figures on the number of deaths from diseases of the coronary artery previous to 1930, the writer has reclassified according to the 1930 International Classification the causes of all the deaths from heart disease occurring in Brookline, Massachusetts, since 1900. The causes of death are those given on the death certificates by the attending physicians or the medical examiners—the latter signing many of the certificates when death was sudden. The figures obtained are of course no more accurate than the physicians' diagnoses.

Brookline had a population of 19,935 in 1900, and this increased to 50,319 in 1935. Women predominate; in those over forty-five the ratio was sixty-one to thirty-nine in 1930. The population shows some ageing. The United States Census Report for 1900 does not give the population of Brookline by ages but in 1910 the Report showed 25.5 per cent of the population over forty-five; in 1920, 30.9 per cent; in 1930, 33.4 per cent. This amount of ageing is not sufficient to explain either the increase of over 100 per cent in the total death rate from all forms of heart disease during this period or the very great increase in coronary disease which this study disclosed.†

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†The writer is indebted to Miss Ruth Coughlin and the F. E. R. A. for much valuable assistance in this work during 1934.

RESULTS OF THE STUDY

The deaths from heart disease have been divided into five groups as follows:

1. Endocarditis, acute and chronic (91 and 92).
2. Myocarditis, all forms (93).
3. Other diseases of heart (95).
4. Angina pectoris (94a).
5. Disease of Coronary Artery (94b).

The rates of each of these groups per 100,000 population in five-year periods were obtained and are to be found in table 1. The figures for 1935 have also been added. These rates are also given in the accompanying chart, frequent reference to which and familiarity with the numbers used in the International Classification will make clearer the explanation in the text that follows.

TABLE 1

DEATHS FROM HEART DISEASE IN BROOKLINE 1900-1935

Rates per 100,000 for the Different Forms and for All Forms

Period of Years	Endocarditis	Myocarditis	Other Diseases	Angina Pectoris	Coronary Disease	All Forms
	91-92	93	95	94a	94b	
1900-1904	51.1	24.1	62.3	16.7	0	154.2
05- 09	93.5	33.8	47.1	22.8	3.9	201.2
10- 14	97.1	56.0	47.1	24.5	4.1	228.9
15- 19	67.6	89.5	23.8	28.2	9.9	219.0
20- 24	66.6	115.1	28.9	27.3	13.7	251.7
25- 29	56.4	127.4	36.4	36.4	39.9	296.7
30- 34	46.3	117.1	26.6	22.9	94.6	307.4
1935	23.8	93.1	31.7	21.8	156.6	327.0

All Forms. For all forms combined, the chart shows a progressive increase from a rate of 153.3 per hundred thousand in 1900-1904 to 327 in 1935 except for a slight decline in 1915-1919 which was probably caused by the influenza epidemic, many heart cases having been carried off by that disease.

Endocarditis. In the chart all forms of endocarditis, acute (91) and chronic (92), including all forms of valvular disease, are grouped together. There was an increase for the first fifteen years and since then a very definite decline. This decline appears in all the recent statistical reports and is probably a real one.

Myocarditis. The diagnosis "Myocarditis" appears to be used by physicians for those cases where, in the absence of valvular disease, the

prominent symptom is the failure of the heart muscle. From the point of etiology, therefore, the cases included in this group are of a number of different types. The most important is doubtless that of hypertensive heart disease, where, according to White[1], the heart muscle shows no inflammatory or degenerative changes and hence should not be diagnosed as myocarditis but as "hypertensive heart disease" (95).

Next in frequency to hypertensive heart disease there is included under the heading "Myocarditis" a group of cases where the heart muscle weakens as a result of areas of fibrosis caused by narrowing or occlusion of the coronary branches supplying these areas. Thus there are many cases diagnosed as myocarditis where the primary cause is disease of the coronary arteries. We need to bear this in mind when we are estimating the frequency of coronary disease.

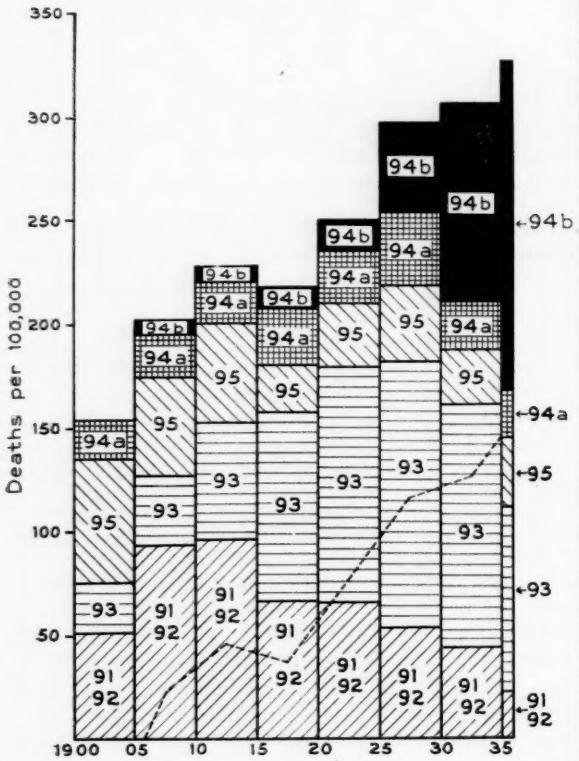

CHART.

Deaths in Brookline, Mass., per 100,000 Population from Different Forms of Heart Disease, 1900-1935.

Endocarditis (91, 92), Myocarditis (93), "Other Diseases" (95), Angina Pectoris (94a), Diseases of the Coronary Artery (94b).

The dotted line indicates the rates for all other forms of heart disease combined if the 1935 rate for coronary disease and angina had prevailed since 1900.

The chart shows that myocarditis (93) increased from a rate of 24.1 in 1900-1904 to 127.4 in 1925-1929, since when there has been a slight decline.

Other Diseases (95). Under this heading are included among others "Cardiorenal disease," "Arteriosclerotic heart disease," and "Cardiovascular disease." The latter is a noncommittal term often used by medical examiners in cases where persons are found dead in their beds or in other cases of sudden death. Certainly the great majority of these sudden deaths are due to coronary disease. The term "arteriosclerotic heart disease" is an unfortunate one, because it is used for two different forms of heart disease: 1. It is used where there is a generalized arteriosclerosis with renal involvement and hypertension; 2. it is used for arteriosclerosis of the coronary arteries. We must remember then that included in "other diseases" (95) are many cases of coronary disease.

Angina Pectoris (94a). The rate for deaths certified as angina pectoris has remained fairly constant during the whole period. It was evident that in the earlier years many cases that would now be diagnosed as coronary thrombosis (94b) were called angina (94a). Thus it was repeatedly found that in cases certified as angina the duration was put down as from a few hours to several days—certainly very typical of coronary thrombosis. Furthermore it was noticeable that when a given physician had once used the term "coronary thrombosis" the diagnosis of "angina" seldom or never again appeared on his certificates.

Apparently the diagnosis "angina" is now used chiefly for those cases where there is a history of previous attacks and death is almost instantaneous. According to White[2], disease of the coronary artery is present in 95 per cent of cases of angina. It is obvious that angina and diseases of the coronary artery should be considered together in any etiological study.

Disease of the Coronary Arteries (94b). It was realized before this study was undertaken that the rates for coronary disease would be profoundly influenced by the fact that only during recent years have physicians recognized coronary thrombosis ante mortem. Herrick's[4] epoch-making paper in 1912 first brought to the attention of the physicians in this country the clinical picture of the disease which is so frequently recognized now, but this knowledge spread slowly to the general practitioners, so that only during the past ten years have any considerable number of physicians been making the diagnosis of coronary thrombosis and their number is still increasing.

In the period 1900-1904 the word coronary does not appear on a single death certificate. In the four periods from 1905 to 1924 the rate for coronary disease increased from 3.9 to 13.9 but jumped to 39.9 in 1925-1929. From 1930 to 1936 the increase has been very rapid, the rates for these six years being: 58.7; 76.6; 94.1; 101.2; 140.0; and in 1935, 156.6.

The high incidence of coronary disease in the Jewish race was obvious. Thus during 1934

and 1935 of all men dying from this cause 18.8 per cent were born in Russia, almost all of whom were probably Jews. Among men dying of all other forms of heart disease except endocarditis only 1.4 per cent were born in Russia.

The study also showed the high incidence of coronary disease among business and professional men. Thus of 489 men dying of coronary disease and angina 67 per cent were of the business and professional class, while of 704 men dying of myocarditis (93) and "Other forms" (95) only 48.7 were business and professional men, despite the fact that the ages of the two latter groups were higher.

The age distribution of coronary disease among men and women is shown in table 2. From this it appears that women die at an older age than do men. Among women 48.8 per cent were below seventy, while among men 66.7 per cent were below seventy.

TABLE 2

CORONARY DEATHS IN BROOKLINE 1900-1935 ACCORDING TO AGE AND SEX

	Total Deaths	% in Each Age Group 20-44	45-59	60-69	70-
Men	299	7.4	26.8	32.5	33.3
Women	162	1.9	9.9		51.2

The rates for coronary disease would have been much higher, were it not for the fact that in Brookline women predominate, sixty-one to thirty-nine. Computing the rates for each sex separately and for the ages over forty-five, we find that in 1935 the rate was 811 per 100,000 men over forty-five, and for women, 333.

HAS THERE BEEN A REAL INCREASE IN CORONARY DISEASE?

Obviously a large part of the increase of coronary disease noted in the death certificates is due to the fact that physicians are now diagnosing more cases of coronary thrombosis than formerly, but does this explain all the increase? Can we find any evidence in the figures that have been collected in this study to show that there has been an actual increase?

If there has been no real increase (or decrease) then the 1935 rate per 100,000 population, 178.4 for angina and coronary disease combined, must have prevailed during the whole thirty-six years under consideration, and the cases not diagnosed as such must have been diagnosed as some other form of heart disease and have been included in other subdivisions. We are probably justified in assuming that very few were diagnosed as endocarditis (91 and 92). The majority must then have been included under myocarditis (93) or "Other diseases of heart" (95).

In the chart the dotted line indicates the total of all other forms of heart disease if the 1935 rate for angina and coronary disease prevailed throughout the whole period. It shows that the present rate for coronary disease is greater than the rate for all forms of heart disease in 1900-1904, and only slightly less in 1905-1909. It is therefore impossible that the present rate prevailed then. In the next two periods, 1910-1919, if the present rate prevailed there could have been no deaths from Myocarditis (93) or "Other diseases" (95). During the next three periods, 1920-1934, if we assume that the 1935 rate prevailed, then we must also assume that during that period there has been an enormous increase of myocarditis and "other diseases", an increase for the two combined from a rate of 6.6 in 1920-1924 to a rate of 137.1 in 1935. This would be a very difficult increase to explain.

It may be that all forms of heart disease except endocarditis have increased during this period. That there has been a very definite increase in coronary disease the figures here presented certainly show. We must also realize that the number of deaths from coronary disease are much greater now than even the 1935 figures show, for as already pointed out there are still many deaths classified as myocarditis (93) and "other diseases" (95) which are really due to coronary disease. If these could all be included it is possible the rate of 811 per 100,000 men over forty-five in 1935 might be increased to 1000.

When we find that so many of these deaths are of persons in the prime of life—in men 66.7 per cent under seventy years of age—we must recognize that we have here a very large and important problem in preventive medicine toward the solution of which no helpful measures have as yet been suggested.

The first step in the solution of this problem is to determine why arteriosclerosis, a process to be expected in the arteries in old age, attacks the coronary arteries in certain persons at a relatively early age.

WHY THIS INCREASE OF CORONARY DISEASE?

The importance of this question is very generally recognized, and in the literature there is much discussion of it. Without attempting to review this it may be said in summary that most authors attribute the increase in coronary disease to worry and to the stress and strain of modern life—that is to a nervous and emotional strain. Thus White[3] finds the increase in coronary disease "appalling" and believes "the most effective move we can make is to call a halt on the world's mad rush of today". Dr. W. J. Mayo[5] has picturesquely compared the belief of the Ancients that the heart was the seat of the emotions with the modern idea that stern control of the emotions affects the coronary arteries of the heart. He points to the frequency of coronary deaths among surgeons who as a class must have stern control of their emotions.

Osler[6], in his textbook, says of Angina Pectoris: "It is not a disease of the working classes. The life of stress and strain, particularly of worry, seems to predispose to it, and this is perhaps why it is so common in our profession."

Many others besides Osler have called attention to the relative freedom from coronary disease of those doing manual work, but no explanation for this striking fact seems to have been offered. The obvious and striking difference between those groups of society who suffer least and those who suffer most has been largely ignored. This difference is that those who suffer least have occupations which entail a certain amount of *daily* physical activity, while those who suffer most have sedentary occupations which require a minimum of physical activity.

Granting that in those with sedentary occupations nervous and emotional strain may be a contributing factor, *is it not the sedentary life, the insufficient muscular activity, which is the basic underlying cause?*

If physical inactivity is an important factor in the development of coronary disease, then it would be logical to seek the cause of the increase in coronary disease in changes in the habits of the American people which had resulted in lessened physical activity and more sedentary living. On very brief consideration it will become apparent that such changes have taken place.

LIFE IN A MOTORIZED AGE

First and foremost is the use of the automobile. Everyone old enough to remember the "horse and buggy" age must realize what a tremendous change has come about. Almost everyone of the business and professional group walked more then than they do now. There was the walk to and from the electric car or train on the way to business. One did errands on foot—now one takes the auto to go half a block. Even social engagements in the evening meant some walking while now one goes from door to door in the car. People took walks in the country as a pastime, while now we "go places" in the car.

The medical profession was the first to use the automobile in a business way and has thus been longest exposed to its influence. At the present time medical men seldom use their legs except to follow a golf ball. It does not seem to the writer that the practise of medicine today entails any more strain than it did twenty-five years ago, and we cannot explain the increase of coronary disease in our profession in that way. The striking change in this period is the lessened physical activity of men.

In addition to the automobile there are various other labor-saving devices which have resulted in less physical effort. Elevators are in more general use in stores, office buildings, and apartment houses. Oil heaters make unnecessary the shoveling of coal; life in apartment houses reduces the number of chores to be done, such as shoveling snow and caring for the garden. All in all, life for those with sedentary occupations has become much more sedentary.

There is reason to believe that walking is an especially good form of exercise for the heart. In the German spas which have a world-wide reputation for the treatment of heart disease, graduated walking exercises have been used extensively in rehabilitating patients with damaged hearts. Paths are laid out and marked with different colors indicating the steepness of the grades on these paths. The grades to be climbed are carefully prescribed and before discharge the patient is often able to do considerable *"hill climbing"*.

In general, one is safe in assuming that those hygienic measures that are of value in the treatment of a disease will also be useful in the prevention of that disease. Thus sunlight will not only cure but also prevent rickets. The hygienic measures used in the treatment of tuberculosis are also applicable for the prevention of that disease. By a similar analogy walking should be of value for preventing those forms of heart disease which are not of infectious origin.

GOLF AND CORONARY DISEASE

Apparently counteracting the lessened physical activity resulting from the use of the automobile has been the growing popularity of the game of golf among just that class of society that suffers most from coronary disease. One often sees reference in the literature to the frequency of coronary disease among golfers. Riesman and Harris[7] state: "The majority of coronary individuals are active and athletic, often passionate golf players." At first sight this seems inconsistent with the theory that physical inactivity favors the development of coronary disease. We must remember, however, that golf is not *daily* exercise. It is usually played only once or twice a week for a period of about six months and there follows an equal portion of the year when, with little or no reduction in the diet, the average golfer takes little or no exercise.

Golf should be an ideal form of exercise for middle-aged men and doubtless would be if golfers took regularly some other form of exercise—preferably walking—when they were not playing golf. It is obvious that playing golf for six months and then taking no exercise for six months is no protection against coronary disease and may perhaps promote it.

THE PREVENTION OF CORONARY DISEASE

With nervous and emotional strain, the only generally recognized causative factor in coronary disease, preventive medicine has been pow-

erless. We cannot "call a halt on the world's mad rush of today". We have seen our most useful citizens carried off by this disease in increasing numbers and have not raised a finger to prevent others meeting the same fate.

While we can do little to reduce worry and nervous strain, *we can control our physical activities.* Once it is recognized that coronary disease results in part at least from lack of muscular activity we have a very definite point of attack. Obviously the need is for a campaign of education to teach the public (and the profession) that *daily* exercise is one of the essentials of health; that strenuous exercise taken occasionally or limited to only part of the year may be dangerous. Exercise should be a part of our daily routine.

The time is ripe for such a campaign of education, for people are now really alarmed by the frequency of deaths from this cause and would grasp at any practical means of lessening the danger. The tuberculosis campaign has shown us what may be accomplished by education. As a problem in preventive medicine, coronary disease is now much larger and more important than is tuberculosis. It is second only to cancer.

It is certainly the duty of the medical profession and especially of the cardiologists, to consider seriously the question whether the lessened physical activity of the present day is not an important cause of the increase of coronary disease. If they find it is, the course to be followed is clearly marked.

SUMMARY

Previous to 1930 there was no subdivision for coronary disease in the International Classification of Causes of Death. To make figures available for coronary disease previous to 1930 the writer reclassified, according to the 1930 Classification, all the deaths from heart disease in Brookline from 1900 to 1935.

There was a very marked increase of coronary disease during this period. In 1935 the rate for men was 811 per 100,000 men over forty-five, and for women of the same ages, 333. Actually the rate is even higher as there are still many deaths due to coronary disease not diagnosed as such.

A part of the increase noted is due to the fact that coronary thrombosis is more frequently diagnosed now than formerly, but this study showed that there must actually have been a very considerable increase.

The early appearance of sclerosis of the coronary arteries in certain individuals is most commonly attributed to nervous and emotional strain. Little attention has been paid to the fact that the disease is rare in men with occupations requiring daily physical effort and most frequent in those with sedentary occupations, which suggests that physical inactivity predisposes to coronary disease.

The life of men with sedentary occupations has become even more inactive through the use of the automobile and other modern devices, and this may well explain in part the recent increase in coronary disease.

To prevent coronary disease a campaign of education is needed to teach the American people that *daily* exercise is one of the essentials of health.

REFERENCES

1. White, Paul D.: Heart Disease. New York: The Macmillan Company. P. 393, 1934.
2. Idem: P. 610.
3. Idem: P. 609.
4. Herrick, J. B.: Clinical features of sudden obstruction of the coronary arteries. J. A. M. A. **59:** 2015 (Dec. 7) 1912.
5. Mayo, W. J.: Remarks on romance of medicine. Proc. Staff Meet. Mayo Clin. **10:** 393 (June 19) 1935.
6. Osler, Sir William: The Principles and Practice of Medicine. Eighth Edition. New York: D. Appleton-Century Company, Inc. P. 837.
7. Riesman, D., and Harris, S. E.: Disease of coronary arteries with consideration of data on increasing mortality of heart disease. Am. J. M. Sc. **187:** 1 (Jan.) 1934.

A CLINICAL AND PATHOLOGICAL STUDY OF ONE HUNDRED AND FIFTY CASES OF TUBAL PREGNANCY*

BY BENJAMIN TENNEY, JR., M.D.†

A STUDY has been made of one hundred and fifty cases of tubal pregnancy which have been treated on the Gynecological Service of the Boston City Hospital during the past eight years. The hospital records of the patients, the laboratory work, and the pathological specimens in the Department of Pathology have been studied. From the data available as complete a picture as possible has been drawn.

In regard to menstrual history, out of one hundred and forty cases whose histories were satisfactory, sixty-four or 45 per cent gave the story of having missed one period entirely. Eight had missed two periods and one had missed three. Therefore, seventy-three or 52 per cent had missed one or more periods. Add to this, twenty-five cases that had a very scanty period with just a little spotting and we have a total of 70 per cent. Jonas' figure of 60 per cent is quite close[1]. A history of irregular bleeding previous to admission was given by one hundred and sixteen or 82 per cent. There were one hundred and sixteen or 77 per cent of the hundred and fifty cases that were bleeding on ad-

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mission. These last figures seem of especial interest as it stands out definitely that the acute type of cases and the more dangerous were those that did not show staining on admission. They were the type that had been well until seized with acute abdominal pain generally within twelve hours of admission. In this class fall most of the severe ruptures with internal hemorrhage. The absence of bleeding indicates a living pregnancy up to the time of rupture and therefore a more potentially dangerous source of disaster. Three of the five deaths from acute hemorrhage were not staining on admission.

The physical findings in the entire series gave one hundred and forty or 93 per cent whose chief complaint was lower abdominal pain usually worse on the side of the pregnancy but in many it was bilateral. Abdominal tenderness was present in one hundred and twenty-six or 84 per cent. Pelvic tenderness either localized or general was present in one hundred and thirty-nine or 92 per cent. A pelvic mass was made out in ninety-one or 60 per cent. There were forty-two cases or 28 per cent in which although a definite mass could not be outlined, there was a notation of bulging or fullness. In other words there were only seventeen or 12 per cent that did not show either a pelvic mass or fullness. An abdominal mass was palpated in eleven cases.

Out of the entire series twenty-six cases or 17.3 per cent were primiparas. In eighty cases in which the history was complete in this respect, thirty-eight had had a pregnancy within two years, nineteen within five years and twenty-three from five to twenty-five years. A long period of sterility was not the rule in this series. It was interesting to learn that forty-eight cases or 32 per cent had had previous abdominal operations of which all but seven were appendices or pelvic surgery. This point has been emphasized by Litzenberg[2] who however has the lower figure of 15 per cent. Scheffey, Morgan and Stimson[3] give 37.7 per cent which is close to our figures. Only eighteen cases had a past history of pelvic inflammation but as that is a difficult history to obtain, I feel it fairer to rely on the pathological sections which show a much higher incidence. Litzenberg[2], however, finds in his pathological sections only 10 per cent definite salpingitis.

Correct preoperative diagnosis was made in 79 per cent. Fifteen cases were diagnosed as pelvic inflammation, seven cases as ovarian cyst, three cases as appendicitis, three as fibroid uterus, and two as pelvic abscess with threatened miscarriage. In eight cases no definite preoperative diagnosis was recorded.

LABORATORY

There were ninety-one cases in which a white blood count was recorded. Of these, forty-seven or a little over half were under ten thousand. There were nineteen cases between ten and fifteen thousand. There were twenty-five cases in which the white count was higher than fifteen thousand. Of these twenty-five, twenty-one were of the acute type with symptoms of less than twenty-four hours' duration. Of these twenty-one all showed general abdominal tenderness and spasm and all had considerable free blood in the abdomen at the time of operation. The majority of these were not bleeding on admission. In other words, the high white count indicated acute rupture with internal hemorrhage of considerable amount and of recent origin. The other four cases had had symptoms from one week to a month with some staining on admission. However, they showed general abdominal tenderness and spasm with fresh blood in the abdominal cavity, in other words an acute hemorrhage on top of previous minor bleeding.

Of particular interest is the sedimentation rate that was done on thirty-six cases. There were twenty-two cases that had a sedimentation time of one hour or greater. Eleven cases were between thirty minutes and one hour. There were only three cases under thirty minutes. This certainly indicates that the sedimentation rate is a valuable point in differential diagnosis of pelvic infection and hemorrhage from tubal pregnancy. Pelvic inflammation with symptoms sufficiently acute to be confused with ruptured tubal pregnancy usually shows marked lowering of the sedimentation rate.

An Aschheim-Zondek test was done in eighteen cases. In the case of acute rupture with internal hemorrhage there is not the time or need for such a test. The diagnosis is usually fairly definite and if not positive, at least shows an acute abdominal condition that needs surgery. In the case with pain and a pelvic mass but no acute symptoms, the Aschheim-Zondek test is of immense value. In spite of the small number of tests, the definiteness of the findings was of considerable value. Of the eighteen cases eight tests were positive. In the microscopic study of these every one showed actively growing trophoblast (fetal cells). In three cases the placental villi were necrotic or absent. This coincides with the belief as shown in hydatid mole and chorio-epithelioma that the hormone is dependent on the trophoblast and not the fetus. In two cases with sections of the ovary a good corpus luteum was found. In the other ten cases the Aschheim-Zondek test was negative. In these ten cases no living trophoblast could be found in any of the sections. The sections generally showed organized blood clot containing hyalinized placental villi and occasional trophoblast cells in a degenerated and necrotic state. In six cases there were sections of the ovary and in none was there any trace of the corpus luteum of pregnancy. The value

of the Aschheim-Zondek test appears to be this. If the test is positive, it indicates that there are living, growing and invading fetal cells present which give a definite danger of rupture of the tube, of hemorrhage or of both. On the other hand if the test is negative, the danger is less acute with a much slighter chance of internal hemorrhage. It is also important to note that a negative Aschheim-Zondek test simply excludes a living tubal pregnancy and does not rule out a dead tubal pregnancy as the explanation for what symptoms may be present.

PATHOLOGY

In reviewing the reports on the gross pathology of the specimens it is quite difficult to classify the cases as rupture or abortion. However, there were seventy-nine cases or 52 per cent that showed definite perforation or rupture of the tubal wall. There were thirty-four cases described as intact. By this is meant cases in which there was no perforation of the tubal wall and no evidence of extrusion of tubal contents from the tubal ostium. There were thirty-seven cases with no sign of perforation of the tubal wall and evidence of extrusion of tubal contents from the ostium. I agree with Litzenberg[2] that a true and complete tubal abortion is rare. As shown by the microscope this type is practically always an incomplete tubal miscarriage. I also consider his term "internal rupture" excellent as it is rare not to find bleeding within the tube in the intact cases and the so-called abortions are simply a continuation of this process.

Out of the one hundred and fifty cases there were twenty embryos recovered including one pair of twins apparently of a single ovum, each twin being about 2 cm. in length. The embryos generally were found in the intact cases but some of them were recovered from the abdominal cavity following rupture. There was one interesting case of essentially an abdominal pregnancy where the placenta was attached to the appendix and cecum with the fetus growing free in the abdominal cavity. The fetus was approximately three months' size and well formed. There were two cases in which the patient claimed to have miscarried a fetus from the uterus previous to entry. In one case the fetus had been seen by the attending doctor. One certainly and both possibly were the rare condition of a double pregnancy.

The question of salpingitis as an etiological factor has been much discussed. Litzenberg[2] gives his findings as 10 per cent positive and 10 per cent questionable; Scheffey, Morgan, and Stimson[3] as 30 per cent, and Van Etten[4] as 10.4 per cent, Sampson[5] 36 per cent. In this series of cases a somewhat higher incidence was found. In the routine pathological reports thirty-eight cases or 25 per cent were diagnosed as salpingitis. In reviewing the slides with this point in view I found fifty cases or 33 per cent with definite follicular salpingitis. There were two cases that showed tuberculous salpingitis.

In estimating the number of pregnancies that were living at the time of operation ninety-six or 64 per cent showed living fetal trophoblast and fifty-three showed no living fetal cells.

There were forty cases in which specimens of ovary were present. It was interesting to study the relation between trophoblast and the corpus luteum. In sixteen cases the pregnancy was dead with no living fetal tissue and no corpus luteum of pregnancy. The ovaries showed ripening follicles and four had a corpus luteum of menstruation. In eleven cases the pregnancy was in good condition with actively growing fetal tissue and a good corpus luteum of pregnancy. In four cases there was good trophoblast, no good fetal villi and degeneration of corpus luteum. In five cases there was an entirely degenerated corpus luteum and no living fetal tissue. In four cases there was good fetal tissue but no corpus luteum. Of course here the corpus might have been in the other ovary. It would seem from these studies that injury to the pregnancy causes first the death of the fetus. Death of the fetus is followed by degeneration of the corpus luteum. After the degeneration of the corpus luteum the trophoblast may continue to grow for a considerable time. This suggests a hormonal influence of the fetus on the corpus luteum. As we know that removal of the corpus luteum early in pregnancy causes miscarriage, it would seem that the life of the fetus is dependent on the corpus luteum and from the above that the corpus luteum depends on the fetus. Börner[6] found that the corpus luteum begins to degenerate as soon as implantation of the ovum is disturbed.

The "Influence of Ectopic Pregnancy on the Uterus" is well described by Sampson[5]. He found that only the cases of tubal pregnancy that did not have vaginal bleeding had intact uterine decidua. The termination of the pregnancy was based on the first attack of pain but termination was rarely complete at the time of operation. It would seem probable that the action of the tubal pregnancy on the uterine decidua was indirect, acting through the corpus luteum. The uterine bleeding is the sign of beginning degeneration of the corpus luteum caused by death of the tubal embryo. In this series there were six cases in which the uterus was removed. In two cases the tubal pregnancy was intact, the corpus luteum in good condition and the uterine decidua uninjured. In the other four cases there was no section of the ovary. However, in these cases there was considerable living trophoblast and villi in good condition. In one the endometrium was in the normal resting state and in the other premenstrual. In the third the endometrium was atrophic with uterine fibroids and in the fourth the decidua had mostly been cast off. These four cases had been

bleeding for several days. These few cases show that the original injury to the pregnancy causes beginning degeneration of the corpus luteum followed by uterine bleeding and that living trophoblast can survive for some time after.

In the entire series there were six deaths or a total mortality of 4 per cent. One case entered before operation with a temperature of 101°. She died eleven days postoperative of peritonitis and bronchopneumonia. Undoubtedly her death was primarily of infectious origin. The other five were all due to internal hemorrhage. Two of these had acute rupture with a three months' fetus found in the abdomen and died within a few hours in spite of all treatment. The third was a similar type with a four cm. fetus. The fourth was so severely exsanguinated that she died on admission. The fifth was of interest in that she had been under observation for four days. Operation was decided on and she was given 500 cc. of blood before operation. Immediately following the transfusion she went into shock. She was operated on immediately. The abdomen was full of fresh blood and she died on the table. This is a good example of the danger of transfusion starting a fresh hemorrhage.

SUMMARY

A study has been made of one hundred and fifty tubal pregnancies. The outstanding signs and symptoms of these cases were: seventy per cent had either missed a period or had a very scanty period. Eighty-two per cent gave a history of irregular bleeding. Seventy-one per cent were bleeding on admission. Some of the most acute cases were those that were not bleeding when seen. Three of the five deaths from hemorrhage were not staining on admission. Ninety-three per cent complained of lower abdominal pain. Pelvic tenderness was present in ninety-three per cent, pelvic mass or fullness in eighty-eight per cent.

A high white blood count indicated recent and considerable internal hemorrhage. The sedimentation rate in all but three cases was higher than thirty minutes. A positive Aschheim-Zondek test showed living fetal tissue that was still growing and invading. A negative test indicated that growth had ceased.

A study of pathological sections showed that injury to the pregnancy caused first the death of the fetus followed by degeneration of the corpus luteum. The trophoblast often continued to live for some time. The possibility of an hormonal influence of the fetus on the corpus luteum is suggested. Bleeding from the uterus is dependent on beginning degeneration of the corpus luteum which follows the death of the fetus.

CONCLUSIONS

1. The most consistent picture of tubal pregnancy is a history of a missed or scanty period, irregular bleeding, lower abdominal pain and pelvic mass or fullness, and pelvic tenderness.
2. Lack of staining may be a sign of danger.
3. A high white count indicates considerable internal bleeding. A normal sedimentation rate is in favor of tubal pregnancy.
4. A positive Aschheim-Zondek test indicates living fetal cells. A negative test shows death of fetal cells.
5. A study of the pathological specimens indicates that the fetus dies first, followed by degeneration of the corpus luteum. Degeneration of the corpus luteum causes casting off of uterine decidua with uterine bleeding. The trophoblast can continue to grow for some time longer.

REFERENCES

1. Jonas, A. F., Jr.: An evaluation of signs and symptoms in the diagnosis of extra-uterine pregnancy. New Eng. J. Med. **209:** 1324 (Dec. 28) 1933.
2. Litzenberg, J. C.: Tubal pregnancy. Nelson Surgery **8:** 497.
3. Scheffey, L. C.; Morgan, T. R., and Stimson, C. M.: An analysis of a series of 82 cases of ectopic pregnancy. Am. J. Obst. & Gynec. **24:** 103 (July) 1932.
4. Van Etten, R. C.: Is salpingitis a factor in the incidence of tubal pregnancy? Am. J. Obst. & Gynec. **22:** 645 (Oct.) 1931.
5. Sampson, J. A.: The influence of ectopic pregnancy on the uterus, with special reference to changes in its blood supply and uterine bleeding. Based on the study of 25 injected uteri associated with ectopic pregnancy. Surg. Gynec. & Obst. **18:** 587 (May) 1914.
6. Börner, R.: Der anatomische Befund am Uterus als Ausdruck innersekretorischer Funktion bei lebender und abgestorbener Tubargravidität. Ztschr. f. Geburtsh. u. Gynäk. **101:** 763, 1932. See Ref. No. 2.

CONGENITAL ABSENCE OF THE VERMIFORM APPENDIX IN A PATIENT WITH MENTAL DISEASE

BY L. W. DARRAH, M.D.*

CONGENITAL absence of the vermiform appendix is exceptionally rare. According to the ultracritical reviewers of the literature on this subject, there are, seemingly, but twenty-eight authentic reported cases in the past two hundred and sixteen years! Other reviewers of this same literature, and who are just as earnest as the ultracritical group, would extend this number to forty-nine. Therefore, because of such evident rarity, the following case found at autopsy—with no record or evidence of any operation—merits reporting.

Female, aged fifty-five; a patient in the Gardner State Hospital during the past nine years was diagnosed, schizophrenic, paranoid type. She was quite debilitated both men-

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tally and physically and died of gastric ulcer.

At autopsy, June 20, 1935, there was nothing significant in the thorax, or in the abdomen, except in the gastrointestinal tract. The stomach was very white and markedly distended, and when opened, it contained a large amount of gas and a small quantity of grayish-brown fluid. A perforating ulcer 2.5 cm. in diameter was found on the posterior wall, near the lesser curvature. The large bowel, especially the sigmoid, was distended and dark in color. The appendix could not be located. The anterior tenia, said to be the surest guide to the process, was followed as in Green and Ross's case[1] . . . "to its termination, at which point the cecum was smooth. The point of junction of the three teniae was thoroughly examined. Not even a rudimentary projection could be found. The serosal surface of the cecum was everywhere smooth." The retrocecal fold and fossa were also explored. The cecum was the usual lop-sided adult form with nothing else remarkable about its appearance.

It is more than interesting that this rare anomaly was first reported by Morgagni[2] in 1719. Two hundred and sixteen years ago! It was next described by Hunter[3] in 1762 and Haller[4] in 1765. Still later Meckel[5] in 1812; Ferguson[6] in 1891; Roberts[7] in 1896; Dixon[8] in 1896; Swan[9] in 1898; Fawcett and Blachford[10] in 1899; Picquand[11] in 1900; Michaux[12] in 1902; Huntington[13] in 1903; Marie[14] in 1903; Dillard[15] in 1903; Schridde[16] in 1904; Marshall and Edwards[17] in 1906; Looten[18] in 1908-9; Froelich[19] in 1910; Dailey[20] in 1910; Shiels[21] in 1911; Velyaminoff[22] in 1911; Lecompte[23] in 1911; Bloodgood[24] in 1911; Hansen[25] in 1912; Bérard and Buche[26] in 1913; Gladstone[27] in 1914-15; Dorland[28] in 1925; Maurer[29] in 1927; Jacobs[30] in 1928; Bradley[31] in 1929; Spivack[32] in 1931; Green and Ross[33] in 1933; Sindoni[34] in 1933; Louyot, Richon, and Lacourt[35] in 1934; and Darrah in June, 1935.

According to the literature, examined at the Boston Medical Library and the Harvard Medical Library, on congenital absence of the vermiform appendix, the number varies between the ultracritical estimate of twenty-eight reported authentic cases, and forty-nine. Some writers reported two or more cases; some from anatomic and pathologic laboratories, Bird[36], and Bird, Oliver, and Robinson referred to by Dorland[28]. So it is apparent that there is some disagreement, among the reviewers, as to the actual number of authentic reported cases.

Schridde, for instance, when reviewing the subject in 1904, did not accept many of the earlier cases of agenesis of the vermiform appendix. His theory, according to Spivack, is that "the appendicular portion of the primitive cecum fails to become arrested in its development as it normally should, but keeps pace in growing with the cecal portion proper and at maturity is indistinguishable as to caliber from a normal cecum. The reason why he gave such an explanation was that in his case the cecum was of a child fifteen months old; on the cecum were present six haustra instead of four. (As is known, the human cecum up to the age of four or five years has deep furrows which subdivide the wall of the cecum into four haustra; these haustra normally disappear at the age of four or five years, due to intracecal pressure of the fecal material, and the cecal wall then becomes smooth.) The fact that in his case there were six haustra instead of four and also the absence of the appendix, made him believe that these two additional haustra were the modified appendix. He explained, on the basis of his specimen, that normally the appendix is formed by the arresting of the lower two haustra; that at some period of the embryonic stage all six haustra are of the same size and width; later on the lower two haustra are arrested in their growth and form the appendix and the upper four continue to grow and form the cecum. Therefore, Schridde advised: Whenever there is a congenital absence of the appendix, count the number of haustra."

This sounds interesting. But, according to Spivack, in order to corroborate it, one has only to deal with such a cecum where the haustra are present, which occurs only in cases of children up to four or five years. In grown persons there are no haustra, and dealing with an absence of the appendix in a grown person, one can neither corroborate nor disprove this theory unless one comes across a case of absence of the appendix in a grown person with an infantile type of cecum. It seems that Schridde had but one such case, and Spivack had one. Obviously, if the former's theory be accepted, then apparently there are but two authentic reported cases of agenesis of the vermiform appendix!

But it will be seen, further on in this article, that Schridde's drastic reduction of cases is set aside by Bradley's study of the embryological development of the cecum, and it receives the support of reputable anatomists.

Dailey, when examining the literature in 1910, would accept only ten of the twenty-six reported cases up to that time. He cautioned that "one should not be too hasty in publishing alleged cases of absent appendix for there are many possibilities of error". Dorland, reviewing the literature at a still more recent date (1925) accepted thirty-seven cases as authentic. Bradley, in his informative article published in 1929, at that time accepted forty cases. He quoted Kelly and Hurdon[37] who state that the appendix is "merely a portion of the general cecal pouch which has remained in an early stage of development". Writes Bradley: "It is formed from the terminal portion of the cecal pouch from which it is at first indistinguishable. Differentiation takes place in two stages:

A primary stage occurs at about eight weeks when a distinction can first be made between a larger proximal portion (cecum) and a smaller distal portion (appendix). A secondary stage occurs at about the time of birth, coincident with the formation of the teniae, when a more marked disproportion between the size of the cecum and that of the appendix becomes evident. Development may be interfered with at any stage. The resultant anomalies have been postulated (by Bradley) as follows:

THE ARRESTED DEVELOPMENT OF THE CECAL POUCH AND THE RESULTANT ANOMALIES

Stage of Growth	*Anomaly*
Failure of the cecal anlage to develop.	Absence of cecum and appendix.
Partial development of cecal anlage.	Rudimentary cecum without the appendix.
Full development of the cecal portion; no development of the appendiceal portion.	Normal cecum; no appendix.
Normal early differentiation of the appendix, but early discontinuance of development.	Normal cecum; rudimentary appendix.
Normal early development but failure of appendiceal portion to become differentiated, its growth keeping pace with that of the cecal portion so that a differentiation is not evident.	Normal appearing cecum probably with extra haustra, as in case reported by Schridde; no appendix.

In 1930, Spivack skimmed the literature and wrote, "This makes a total of forty-four cases and my cases are the forty-fifth and forty-sixth." Yet Spivack evidently had some uncertainty about his figures, as he observed that some of the reported cases were hypoplasia. Excluding these, and those where the appendix was not found but in which there were adhesions around the cecum, then the cases of actual agenesis of the appendix could be cut to nearly half.

"Probably," wrote Spivack, "it would be closer to the truth to say that only about twenty-five cases were undoubtedly true agenesis."

And in 1933, when Green and Ross studied the literature, they too agreed with Spivack's minimum—twenty-five—to which they added their own, the twenty-sixth.

In December, 1933 Baldwin[38], after reading Green and Ross's report, examined Bradley's, and Spivack's, review, also several older ones, then quoted Scott[39], who boldly asserted in 1897 that the appendix is never absent, and that failures to find it have been due to improper search. According to Baldwin, Scott was then Professor of the Principles of Surgery, in the Cleveland College of Physicians and Surgeons, etc., at the time he published an account of a seeming case with a photograph of the specimen. Scott claimed that his own case, and all other reported cases, were merely appendicitis obliterans totalis. Briefly, he located the appendicular artery and tracing that up found ample evidence of the previous existence of the appendix.

In the literature there seems to be no other reference to Scott's paper, and Baldwin explains that the paper was published in the bound volume of the Transactions of the Ohio State Medical Society for that year (1899), and distributed only to paid-up members, and for that reason was practically buried.

Scott's refutation is interesting. It may be that some of the reported cases did have hidden, microscopic vestiges of the appendix. Yet there is proof and authority for total, complete absence of the vermiform appendix. In 1929 there were four known specimens in museums. (There may possibly be more now.) Two are in the Museum of the Royal College of Surgeons, London; and two in New York's Columbia University Museum.

Anatomists agree that it is possible. Spitzka[40] wrote, "In rare instances the appendix has been absent." Piersol[41] states, "The total absence of the appendix is extremely rare, but has been observed by ourselves and others." And Hertzler[42] goes so far as to say, "No one has ever seen a 'normal' cecum except the one depicted in the textbook of his student days."

As has already been mentioned, Bradley has shown in his interesting table on embryological development of the cecum, that not only can the appendix be absent, but the cecum also. (This was shown by Froelich's reported case[19] in 1910 being in complete agreement with Bradley's study.)

It is true, of course, that cases found at autopsy can be examined far more readily than those found during a surgical operation. To verify, on the operating table, the absence of the appendix in case it is not seen at the place of union of the three teniae, Spivack advises that one should mobilize the cecum from the lateral side and examine its posterior wall. And certainly Green and Ross, in their reported surgical case, searched exhaustively. So have other investigators.

After all, why should there be any doubt as to the possibility of absence of the vermiform appendix? Complete absence of the eyes has been recorded. Also complete absence of teeth from birth till death; also absence of feet; bilateral absence of the external ears; congenital absence of the tongue; of the gallbladder; and but one kidney.

SUMMARY

The writer of this paper, in reporting his own case found at autopsy, records that the entire bowel was carefully dissected out, in an attempt to locate the appendix, or find a vestige of its existence. Five other physicians were present and assisted in the search for the appendix, and were finally convinced that here was a case of total congenital absence of the vermiform appendix.

Therefore, this case, if based on the ultraconservative opinions of Schridde, Dailey, Spivack, and Green and Ross, would be approximately the twenty-ninth. But if based on the figures and studies of Dorland, and of Bradley, it would appear to be the fiftieth.

REFERENCES

1. Green, S. J., and Ross, W. J.: Congenital absence of vermiform appendix. Ann. Surg. **98:** 316 (Aug.) 1933.
2. Morgagni, G. B.: Adversaria anatomica. **3:** 14, 1719. Quoted by Dorland and Dailey. See references 28 and 20.
3. Hunter, W.: Medical Commentaries, London, 1762. Quoted by Dorland. See reference 28.
4. Haller, A. von: Elementa physiologiae. **7:** liber 24, section 3, P. 18. Quoted by Dorland. See reference 28.
5. Meckel, J. F.: Handbuch der path. Anat., Bd. i, s. 599, 1812. Quoted by Dorland. See reference 28.
6. Ferguson, John: Some important points regarding the appendix vermiformis. Am. J. M. Sc. **101:** 61 (Jan.) 1891.
7. Roberts, J. B.: Case of congenital absence of the vermiform appendix and of intestinal diverticulum. Ann. Surg. **23:** 611 (May) 1896.
8. Dixon, A. F.: A rare condition of the vermiform appendix. J. Anat. & Physiol. **31:** 442 (April) 1896-97.
9. Swan, J. M.: A case of congenital absence of the vermiform appendix. Univ. M. Mag. **8:** 194 (Dec.) 1895-96. Trans. Path. Soc., Phila. **18:** 38, 1898.
10. Fawcett, E., and Blachford. J. V.: J. Anat. & Physiol. **34:** 20 (June) 1900. Quoted by Dorland. See reference 28.
11. Picquand, G.: Bull. Soc. Anat. de Paris. **68:** 602 (June) 1900. Quoted by Dorland. See reference 28.
12. Michaux, J.: Absence of vermiform appendix. Virginia M. Semi-Month. **7:** 242 (Sept.) 1902-03.
13. Huntington, G. S.: The anatomy of the human peritoneum and abdominal cavity. Philadelphia: Lea Brothers & Co. P. 249, 1903. Quoted by Jacobs. See reference 30.
14. Marie, R.: Bull. et mém. soc. anat. **5:** 466, 1903. Quoted by Spivack. See reference 32.
15. Dillard, J. W.: Congenital absence of the appendix. Virginia M. Semi-Month. **8:** 418, 1903-04. Quoted by Dorland. See reference 28.
16. Schridde, H.: Über den angeborenen Mangel des Processus Vermiformis. Virchows Arch. f. path. Anat. **177:** 150, 1904. Quoted by Jacobs, Spivack and Dorland. See references 30, 32 and 28.
17. Marshall, H. T., and Edwards, R. I.: Agenesis of the vermiform appendix. Philippine J. Sc. **1:** 1061 (Dec.) 1906. Quoted by Dorland. See reference 28.
18. Looten, J.: Un cas d'absence presque totale d'appendice iléocaecal. Bibliog. anat. **18:** 299, 1908-09. Quoted by Jacobs and Dorland. See references 30 and 28.
19. Froelich, M.: Absence of caecum and appendix. Rev. med. de l'est. **42:** 522, 1910. Quoted by Dorland and Jacobs. See references 28 and 30.
20. Dailey, U. G.: Total congenital absence of the vermiform appendix. Surg. Gynec. Obst. **11:** 413 (Oct.) 1910.
21. Shiels, G. F.: Absence of appendix. J. A. M. A. **57:** 1535 (Nov.) 1911.
22. Velyaminoff, N. A.: Congenital absence of the appendix. Khirurg. Arkh. Velyaminova. **27:** 778, 1911. Quoted by Jacobs and Dorland. See references 30 and 28.
23. Lecompte, M.: De l'absence congénitale de l'appendice caecal; rapport d'un cas nouveau. Nancy, L. Bertrand. 1911. Quoted by Dorland and Jacobs. See references 28 and 30.
24. Bloodgood, J. C.: Arch. gén. de Chir. **7:** 80, 1911. Quoted by Spivack and Dorland. See references 32 and 28.
25. Hansen: Münch. med. Woch., Bd. **59:** H. 50, s. 2735 (Dec. 10) 1912. Quoted by Dorland. See reference 28.
26. Bérard, L., and Buche, A.: La Province méd. **24:** 788 (June 28) 1913. Quoted by Dorland. See reference 28.
27. Gladstone, R. J.: Congenital absence of the appendix of the caecum. J. Anat. & Physiol. **49:** 414, 1914-15. Quoted by Dorland and Jacobs. See references 28 and 30.
28. Dorland, W. A. N.: Congenital absence of the vermiform appendix. Internat. Clin. **4:** 44 (Dec.) 1925.
29. Maurer, Z.: Rare case of absence of appendix. Polska gaz. lek. **6:** 155, 1927. Quoted by Spivack. See reference 32.
30. Jacobs, A.: Anomalies of the appendix with report of a case of congenital absence of appendix (agenesis) and bicornuate uterus with one cervix. Am. J. Surg. **4:** 500 (May) 1928.
31. Bradley, J. I.: Congenital absence of the vermiform appendix. Arch. Surg. **18:** 1904 (April) 1929.
32. Spivack, J. L.: Congenital absence of the appendix vermiformis. Am. J. Surg. **13:** 297 (Aug.) 1931.
33. Same as number 1.
34. Sindoni, M.: Sopra un caso di assenza totale congenita dell'appendice vermiforme e criptorchidia monolaterale. Nota anatomica, Rinasc. med. **10:** 86 (Feb. 15) 1933.
35. Louyot, J.; Richon, J., and Lacourt: Absence congénitale d'appendice caecal chez un foetus hérédo-specifique. Bull. Soc. d'obst. de gynéc. **23:** 656 (Nov.) 1934.
36. Bird, U. S.: J. A. M. A. **57:** 1784 (Nov.) 1911. Quoted by Dorland. See reference 28.
37. Kelly, H. A., and Hurdon, E.: The Vermiform Appendix. Philadelphia and London: W. B. Saunders Co., 1905.
38. Baldwin, J. F.: Is the vermiform appendix ever congenitally absent?—The appendicular artery the Ariadne thread to the appendix. Internat. J. Med. & Surg. **46:** 576 (Dec.) 1933.
39. Scott, N. Stone: Trans. Ohio State Med. Soc., Cleveland, 1899. Quoted by Baldwin. See reference 38.
40. Spitzka, E. A.: Gray's "Anatomy". 18th Edition. Philadelphia: Lea & Febiger, P. 1299, 1910.
41. Piersol, G. A.: Human Anatomy. 7th Edition. Philadelphia: J. B. Lippincott & Co., P. 1665, 1919.
42. Hertzler: Royster's "Appendicitis". New York: Appleton and Co. P. 12, 1927.
43. Gould, G. M., and Pyle, W. L.: Anomalies and Curiosities of Medicine. Philadelphia: W. B. Saunders Co. P. 296, 1900.

NOTE: The writer wishes to express his appreciation to Drs. W. A. N. Dorland and J. L. Spivack of Chicago, and Dr. M. M. Canavan, Curator of the Warren Museum, Harvard Medical School, for assistance in the preparation of this paper.

THE OCCURRENCE OF ALLERGIC REACTIONS IN ARTHRITIC PATIENTS*

BY ALBERT G. YOUNG, M.D.†

FOR several years investigators have speculated on the possible relationship of Atrophic or Rheumatoid Arthritis to a bacterial allergic reaction. Herry[1] in 1915 and later Zinsser and Grinell[2] 1925, and Swift, Derick and Hitchcock[3] in 1928, introduced findings that suggested a bacterial allergic reaction in both rheumatic fever and rheumatoid arthritis. Rackemann[4] in 1933, reviewed the literature and gave an excellent critical résumé of the work bearing on this subject. He also pointed out that in his experience hay fever and asthmatic patients did not show an unusual incidence of arthritis.

The writer in studying arthritis has worked on the allergic hypothesis[5], and in presenting his studies has routinely inquired (while taking the history) into hay fever, asthma, food and drug sensitization, migraine, urticaria and dermatitis. The histories also included records of focal or general infections (past or present), and whether the onset of the arthritic symptoms was associated with infection, overwork, trauma, etc. In so doing I was impressed, first, with the high incidence of clinical allergy reported by my patients; and secondly, that most of them reported the presence of some infection, single or repeated, antedating the onset of the arthritis. Recently I have studied the case histories of 200 private patients with rheumatoid arthritis, in whom the onset of symptoms (as shown by the present illness in the history) was sufficiently acute for the patient to be able to state quite accurately its relationship to infection or other

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contributing factors. These findings have been tabulated and compared with the records of fifty non-arthritic patients, fifty patients with allergic dermatitis and fifty patients with urticaria. In making this comparison the attempt has been made to gain information concerning: (1) Whether arthritic patients show a greater incidence of allergy than the average group of clinical patients; (2) if so, what type of allergy predominates; (3) what is the most common condition associated with the onset of the disease, and (4) what is the incidence of arthritis in patients with allergic diseases.

Because of the work of Rackemann[4], and Harkavy and Hebald[6], who reported a low incidence of arthritis in hay fever and asthmatic patients, I did not study this phase of the problem.

RESULTS

Table 1 shows that of the 200 patients studied, 6.5 per cent had hay fever; 7 per cent had asthma and 1.5 had both conditions so that actually 15 per cent had a present or past history of hay fever or asthma, or both. Urticaria was found in 15.5 per cent and allergic dermatitis in 16 per cent, making a total of 31.5 per cent showing skin allergy.

TABLE 1

INCIDENCE OF CLINICAL ALLERGY IN ARTHRITIS

200 Arthritic Patients	No.	Per Cent		50 Non-Arthritic Patients	No.	Per Cent	
Hay Fever	13	6.5		Hay Fever	4	8	
Asthma	14	7	15	Asthma	3	6	14
Hay Fever and Asthma	3	1.5		Hay Fever and Asthma	0	0	
Urticaria	31	15.5		Urticaria	5	10	
Allergic Dermatitis	32	16	31.5	Allergic Dermatitis	1	2	12

For comparison I then began a search to determine what percentage of non-arthritic patients with a chief complaint other than that of an allergic disease, had hay fever, asthma, urticaria, or allergic dermatitis. Fifty non-arthritic records (table 1) disclosed a past or present history of hay fever in four patients (8 per cent) and asthma in three patients (6 per cent), making a total of 14 per cent as compared with 15 per cent of the arthritic patients; urticaria in five patients (10 per cent) and allergic dermatitis in one patient (2 per cent), making a total of 12 per cent as compared with 31.5 per cent in the arthritic patients.

From these findings it would appear that arthritic patients are more susceptible to skin allergy than the average clinical patient.

The next question was what per cent of patients suffering from allergic dermatitis and urticaria had arthritis. An examination of the records of fifty cases of allergic dermatitis (table 2) or venenata, showed that two patients (4 per cent) had arthritis. None had a question or a past history of arthritis. The records of fifty cases of urticaria showed one case (2 per cent) of arthritis and none with a question or a past history of arthritis. This makes a total of 6 per cent of arthritis among 100 patients suffering from skin allergy, as compared with 31.5 per cent of skin allergy in 200 arthritic patients.

A study of the relation of infection to the onset of the disease was undertaken. Care was taken in each instance to determine when the bacterial infection occurred in relation to the onset of the arthritis. If the upper respiratory, or dental infection, etc., incident was more than eight weeks previous to the onset of the arthritic symptoms the case was not used for this study, since it would be unusual for anyone to attain the age of twenty-five or thirty years without having some upper respiratory or dental infection, to say the least. Care was also taken to rule out upper respiratory or dental infection which occurred after the onset of the arthritis. This does not mean that infections occurring two months prior to the onset of arthritis are not considered as pertinent to the development of the disease, but for the reason stated above it was necessary to draw an arbitrary line if the findings were to have any value in this study. The results showed, that at the time of, or immediately previous to the first symptoms, sixty-eight (34 per cent) had infected teeth, eighty-four (42 per cent) had sore throat, sixteen (8 per cent) had infected sinuses, four (2 per cent) had middle ear infection, one (.5 per cent) had ruptured appen-

TABLE 2

INCIDENCE OF ARTHRITIS IN CLINICAL ALLERGY

50 Urticaria Patients	No.	Per Cent	50 Allergic Dermatitis Patients	No.	Per Cent
Arthritis	1	2	Arthritis	2	4
? of Arthritis	0	0	? of Arthritis	0	0
P. H. of Arthritis	0	0	P. H. of Arthritis	0	0

dix, and two (1 per cent) had a nonspecific prostatitis. This makes a total of 87.5 per cent in whom an active infection was associated with the onset of the disease. In the remaining 12.5 per cent a history of exposure to dampness and cold, overfatigue, trauma, etc., was recorded.

Skin tests on twenty patients to food and bacterial proteins were of little value so far as linking up the skin sensitization with the arthritis. This corresponds with the work of Derick and Fulton[7] who found positive skin reactions to food and bacterial proteins to be as common in adults who were not suffering from arthritis as in the arthritic patients.

One interesting observation was made in this respect. Even in cases where a known food sensitization was present the patients' joint symptoms were not influenced by eating the foods to which they were sensitized. This experiment was repeated several times on about fifteen patients who suffered with a pronounced urticaria or dermatitis following the ingestion of certain foods. At no time did they present any increased objective findings, nor did they complain of increased symptoms during the time of the skin reaction.

DISCUSSION

These results do not strengthen the contention of some investigators who attribute the etiology of arthritis to dietary disturbance. Some of the findings are very interesting in this study. First, the results show an unusually high percentage of skin allergy either past or present in the arthritic patients, whereas, patients suffering primarily from a skin allergy do not show an unusual incidence of arthritis. This indicates that while arthritis appears to predispose to skin allergy, the reverse is not true. And secondly, the incidence of focal infection in relation to the onset of arthritis is too high to be disregarded. It lends strength to the hypothesis of bacterial allergy.

Just why the asthmatic and hay fever patients do not show a high incidence of arthritis may be answered by Rackemann's explanation and also it is pertinent to the findings mentioned above relative to the incidence of skin allergy in arthritis. In discussing Harkavy and Hebald's[4] findings of 2.25 per cent of arthritis among 400 children with asthma, he says: "Evidently the relation of Arthritis to this particular form of allergy is very doubtful. But the word 'allergy' is also used in connection with other reactions which are not immediate but delayed; which give rise not to urticarial wheals, but to areas of inflammation."

He then points out that at least a theoretical relation exists between the two, namely, the work of Zinsser and others indicates that the urticarial reaction typical of hay fever and asthma represents the early phase of the immune process, whereas, the inflammatory tuberculin type of reaction represents the late inflammatory phase. He believes the mechanism in arthritis to be closely related to that in asthma, but that in both conditions the fundamental feature is not the particular agent but the *reaction of the host toward the reagent.*

Rackemann's opinion is further substantiated by the findings of Young and MacMahon[8] in which it was demonstrated that the pathological lesions in arthritis and rheumatic fever were identical, and concluded that: "This complex histological picture is simply one of a nonspecific chronic inflammatory reaction showing regressive, exudative and proliferative changes, but no structural lesions diagnostic of rheumatic infection."

In view of the frequent references made to tuberculin allergy in speaking of arthritis, it is interesting that while making the above study (Young and MacMahon), a section from a case of tuberculosis of the joint was obtained which showed the same microscopic changes that were described in the arthritic and rheumatic patients. I consider this an inflammatory allergic reaction which is not specific for any one organism. This is in keeping with the view expressed by Birkhaug[9] who said, "It seems justifiable to postulate that the exquisite allergy to streptococcal products is resultant from oft-repeated upper respiratory low-grade infections. The question arises whether or not one or several strains of streptococci are responsible for the early sensitization of the individual."

I would carry this hypothesis farther to include prolonged or oft-repeated low-grade infections anywhere in the body. Furthermore, while the streptococci appear to be the most common offenders, we cannot entirely disregard the possibility of other organisms especially the staphylococci and gonococci as sensitizing organisms. It is also important to consider the sensitization as being brought about by the broken-down products of the bacteria rather than by the bacteria *per se.* This idea is consistent with the variations in the results reported on blood cultures in this disease.

SUMMARY

1. In 200 patients with rheumatoid arthritis 15 per cent gave a history of past or present hay fever or asthma, or both; and 31.5 per cent gave a history of allergic dermatitis or urticaria.
2. In fifty non-arthritic patients 14 per cent gave a history of hay fever or asthma, or both; and 12 per cent gave a history of allergic dermatitis or urticaria.
3. In fifty patients with allergic dermatitis 4 per cent had arthritis.
4. In fifty patients with urticaria 2 per cent had arthritis.

5. In 200 patients with rheumatoid arthritis 87.5 per cent gave a history of an active infection or repeated infections just previous to or coincident with the onset of the disease. Twelve and five tenths per cent gave a history of exposure to dampness and cold, fatigue or trauma just previous to the onset of the disease.

The writer wishes to express his thanks to Dr. F. M. Thurmon for the histories of the 100 dermatological cases presented here, and to Dr. Francis M. Rackemann for his helpful suggestions in preparing this paper.

REFERENCES

1. Herry: Quoted by Zinsser, H.: Resistance to Infectious Diseases. New York, The Macmillan Co., 1931, p. 466.
2. Zinsser, H., and Grinnell, F. B.: Further studies on bacterial allergy. Allergic reactions to the hemolytic streptococcus. J. Immunol. **10:** 725 (July) 1925.
3. Swift, H. F., Derick, C. L. and Hitchcock, C. H.: Rheumatic fever as a manifestation of hypersensitiveness (allergy or hyperergy) to streptococci. Tr. A. Am. Phys. **43:** 192, 1928.
4. Rackemann, F. M.: The rôle of allergy in arthritis. New Eng. J. Med. **208:** 1347 (June 29) 1933.
5. Young, A. G.: The use of o-iodoxybenzoic acid in the treatment of chronic arthritis. Wisconsin M. J. **26:** 346 (July) 1927.
6. Harkavy, J., and Hebald, S.: Association of infectious asthma and arthritis from the point of view of bacterial allergy. Arch. Int. Med. **49:** 698 (April) 1932.
7. Derick, C. L., and Fulton, M. N.: Skin reactions of patients and normal individuals to protein extracts of streptococci. J. Clin. Investigation. **10:** 121 (April) 1931.
8. Young, A. G., and MacMahon, H. E.: Chronic proliferative arthritis in patients with rheumatic fever. J. Bone & Joint Surg. **27:** 151 (Jan.) 1935.
9. Birkhaug, K. E.: Rheumatic fever. 3. Skin hypersensitiveness of patients with rheumatic fever and chronic arthritides to filtrates, autolysates and bacterial suspensions of streptococci. J. Infect. Dis. **44:** 363, 1929.

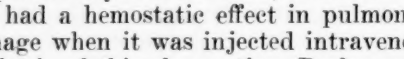
CONGO RED FOR THE CONTROL OF BLEEDING

BY ROGER C. GRAVES, M.D.,* AND C. J. E. KICKHAM, M.D.*

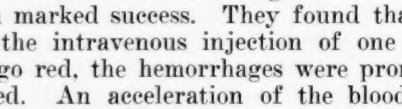
WITHIN the past two years we have had such satisfactory experiences with Congo red for the control of bleeding, that we wish, in this brief communication, to call attention to its value as a hemostatic agent. In cases of active hemorrhage where the ordinary medical measures have failed, it is easily and quickly administered and we have observed no unfavorable reaction to its use, either local or general. The employment of the dye for this purpose seems relatively new and we find that the subject has received but scant mention in the literature.

Wedekind, in 1930, while experimenting with Congo red in an effort to determine the relationship between the storing capacity of the reticulo-endothelial system and the prognosis in pulmonary tuberculosis, observed that the dye had a hemostatic effect in pulmonary hemorrhage when it was injected intravenously. On the basis of this observation, Becker and Wedekind treated fresh bleeding of varied etiology with marked success. They found that following the intravenous injection of one per cent Congo red, the hemorrhages were promptly arrested. An acceleration of the blood clotting time was manifested, due, according to Becker, to the stimulus of the dye solution stored in the endothelial cells of the blood and lymph capillaries. There results an activation of thrombogen and the increased formation of thrombokinase. He observed that both in cases where the coagulation time was retarded, and where it was within normal limits prior to injection, a transitory acceleration took place following injection. In most cases it returned to its previous level within twenty-four hours. When hemorrhages recurred after several days, they were arrested following a single intravenous administration of the dye. It was observed that an increase in the blood platelets accompanied the acceleration of the clotting time. Deinhardt in 1931, studied systematically the influence of intravenous Congo red on gynecological bleeding in a series of thirty cases. The treatment was successful in twenty-two. The hemorrhages in these cases were controlled immediately following the first injection, or after two or three injections on subsequent days.

Congo red is an electronegative dye direct for cotton, that is, it dyes cotton without a mordant, as it also dyes silk and wool. It is turned blue by mineral acids. It is employed commercially in the form of the sodium salt, a reddish-brown powder which dissolves in water to form a red solution. The dye is prepared by diazotizing benzidine



and coupling the diazo compound with the sodium salt of naphthionic acid



(NH$_2$ — α — naphthylamine — p — sulfonate SO$_3$H).

It is used in the medical profession as a test for amyloidosis, and as a means of determining the function of the reticulo-endothelial system. To determine whether Congo red is excreted as such in the urine, 10 cc. was given intravenously to one of the writers. Fifteen and forty-five-minute urine specimens were collected following the injection, and tested to determine the presence or absence of the dye, by Professor Tenney L. Davis of the Massachusetts Institute of Technology. No evidence that Congo red was present was found. This simple experiment suggests, as we would expect from the conclusions of previous writers, that the hemostatic effect is blood-borne, and that the dye does not exert

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any specific effect through the urine when used to arrest hemorrhage from the genito-urinary tract.

Our personal interest in Congo red as a hemostatic agent, is based upon its apparent usefulness in the control of hematuria. We have employed it for the relief of bleeding in such cases as the following: Renal injury; bilateral renal and ureteral calculi; chronic pyelonephritis; vesical calculus; benign hypertrophy of the prostate; tumor of the bladder; urethral trauma, etc. In fact, we have become so convinced of its value that the administration of Congo red is common in our clinic in all cases of active urinary hemorrhage, in conjunction of course with such other specific measures as may be indicated in the particular problem. No blood studies have been made to determine changes in coagulation time or in the platelet count, and we have made no attempt to study the behavior of the dye in the production of hemostasis.

There have been no untoward effects in any instance, following the injection of Congo red in our cases. Five cc. (1 ampule of a sterile isotonic solution) or 10 cc. given intravenously, constitutes the usual dose. This may be repeated if necessary. Rossak states that occasionally patients complain of palpitation and lower abdominal pain, but we have not observed such disturbances. Even the accidental paravenous injection of the dye has resulted in no ill effect, other than a persistent red discoloration of the skin.

In conclusion, it is our feeling that intravenous Congo red is a valuable adjunct in the treatment of hemorrhage from the urinary tract. We believe, however, that it is most useful in cases of acute bleeding in individuals who still possess at least relatively normal coagulation mechanism. We have found it, as might be expected, less effective in such chronic persistent bleeding as often occurs, for example, from pyelonephritis or malignant tumors of the urinary bladder. Whether its lack of success under such circumstances is due to local or general causes, we are unable to state. Obviously it will be of little or no value in actual blood disease. (We have seen it employed without benefit in one case of purpura.) We have used it, frequently, as a prophylactic agent following prostatectomy and transurethral resection, when unusual bleeding had been encountered. It is of interest also, that some of our colleagues have administered it with marked success in such acute conditions as severe epistaxis and hemorrhage after tooth extraction.

REFERENCES

1. Wedekind, T.: Becker, J. and Wienert, B.: Kongorat als Hämostyptikum. München. med. Wchnschr. **77:** 2049 (Nov. 28) 1930.
2. Deinhardt, D.: Behandlung gynäkologischer Blutungen mit Kongorot. Zentralbl. f. Gynäk. **55:** 3249 (Nov. 7) 1931.
3. Rossak, M.: Zur Anwendung des Kongorots bei gynäkologischen Blutungen. Zentralbl. f. Gynäk. **57:** 219 (Jan. 28) 1933.
4. Becker, J.: Blutstillung durch Kongorot; vorläufige Mitteilung. München. med. Wchnschr. **77:** 396 (March 7) 1930.

METHOD OF APPLYING A TEMPORARY ADHESIVE SUPPORT TO THE BACK

BY THOMAS H. PETERSON, M.D.*

IN the past a great deal of unnecessary pain has been produced through attempts to strap or immobilize individuals who are suffering from back pain due to injury or to some other cause. Below is described a method of doing this which was suggested by the late Dr. Robert Soutter and which allows the doctor to apply support to an individual suffering from a very acute back pain without requiring the patient either to turn over in bed, or, as some have been required, to stand. The minimum discomfort to the patient is produced by Dr. Soutter's method and a support is applied which will hold far more firmly than an ordinary binder.

We have had the opportunity of using this type of apparatus frequently in acute back and sacro-iliac strains with good results. It has been used a number of times in separation of the symphysis pubis during pregnancy and in postpartum women. We have also used this type of support several times in fractures of the pelvis, both as a temporary support and also occasionally as permanent supportive apparatus during the healing of the fracture. In its application in separation of the symphysis pubis, whether of traumatic or spontaneous origin, pressure should be applied below the anterior superior spine of the ilium.

A further advantage of this particular method over the usual type of strapping is that it may readily be taken off either during the care of the patient or for the application of physiotherapy, which is often desirable. It is, of course, impossible to apply physiotherapy measures to a back covered with adhesive with much hope of any therapeutic benefits, not only because of the danger of burning, but also there is little chance of effecting results. Since the adhesive strapping on this type of binder is not in contact with the body, no excoriation or abrasion can result from its application and the skin is left in condition for whatever physiotherapy measures are deemed necessary.

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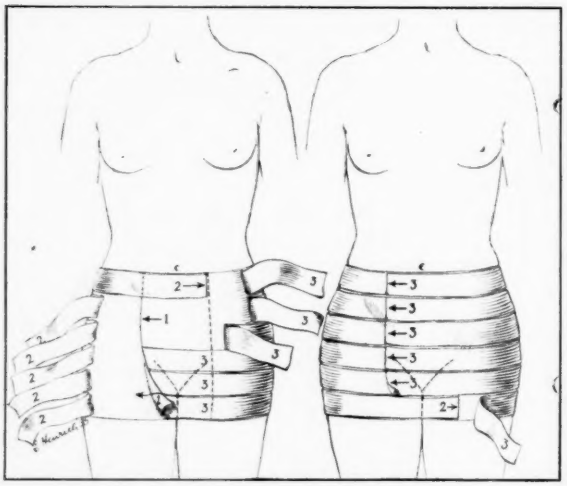
For this method, an old binder or swathe, twelve inches wide, and long enough to encircle the patient with an overlap of about six inches, is procured. To this are applied strips of adhesive tape one and a half inches or two inches wide and of the same length as the binder, put on in parallel lines one eighth of an inch to one fourth of an inch apart, with the adhering side next to the binder. As a general rule six strips are sufficient for the ordinary strapping. We



1. Left end of binder overlaps right end.
2. Right adhesive straps drawn across left end of binder.
3. Left adhesive straps drawn across right adhesive straps.

now have a swathe or binder with five or six adherent straps applied to the outer side.

In applying this support the patient is made to lie on his back and is then gently rolled onto one side. The binder is tucked under the patient's back with the cloth side next to him, he is then rolled onto the opposite side and the binder pulled through and smoothed down flat. The patient is now rolled back so that he is lying flat on his back. The binder with the cloth next to the skin is beneath him, the long axis of the binder at right angles to the long axis of the body and at a level of the greatest discomfort.

Now the adhesive strips are pulled back from either end of the binder for about twelve inches and are laid out flat to be easily picked up again. The two ends of the swathe and binder are pulled across to overlap each other like the overlapping of a double-breasted overcoat (the left end overlapping the right) and are adjusted as snugly as is desirable or as the patient can comfortably stand. The adhesive straps are then brought across from the *right* end so that they now overlap the *left* end of the swathe in front. Each strap is brought across separately and applied to the swathe separately. In this way the necessary adjustments of the swathe may be made so that it will fit smoothly and all loose folds taken up. As a final step the loose end of the strapping, namely the left end, is brought across the now adherent right ends overlapping and adhering securely.

We now have produced a firm binder which fits very comfortably, conforming well to the contour of the body, because of the individual adhesive straps, and applied much more firmly than an ordinary swathe. It has the added advantage of not having injured the skin as the taping never comes in direct contact with the skin.

SUMMARY

This type of strapping is devised for very acute backs and back injuries. It is easily applied to a patient lying on his back with the minimum discomfort. It may be applied more firmly than an ordinary binder. It is easily and comfortably removed for physiotherapy treatment and reapplied with equal simplicity. It does not necessitate the direct application of adhesive plaster to the skin which, in itself, would frequently contraindicate the use of physiotherapy measures.

THE SOCIAL SECURITY ACT

Under the terms of the Social Security Act, the Federal Government matches the expenditures of States with approved plans for assistance to the needy aged and the blind up to a combined total of $30 a month per person and contributes five per cent additional for administrative purposes. For aid to dependent children, the Federal grant to States with approved plans is one dollar for every two dollars the State spends up to a combined total of $18 per month for the first dependent child in any one family and $12 per month for each additional child in the family. Each State decides for itself which of its aged, blind, or its dependent children are entitled to aid and how much aid shall be given.

To be approved by the Social Security Board, a State public assistance plan must provide for cash payments to needy aged persons, to dependent children living with relatives, or to needy blind in all parts of the State. A single State agency must administer the plan or supervise its administration if it is directly administered by the counties. This State agency must grant to any individual denied assistance the opportunity for appeal from the decision of the county denying him such assistance.

Thirty States and the District of Columbia now have public assistance plans conforming with the requirements of the Social Security Act. More than half a million individuals in these thirty-one jurisdictions receive assistance in the form of monthly cash payments under the coöperative Federal-State system of aid provided for in the Act.

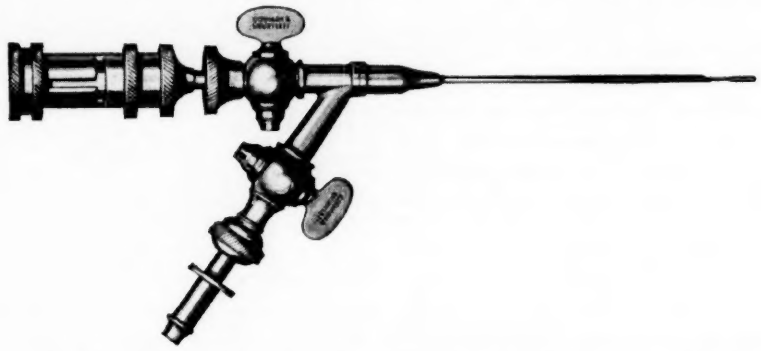
DO YOU KNOW?

Children cry more when the temperature is low, Dr. Catherine W. Brackett, of the Child Development Institute, New York, reports.—*Bulletin, Public Relations Bureau, New York State Medical Society.*

A NEW INSTRUMENT—AN ANTI-ADHESION PNEUMOTHORAX NEEDLE

BY CLEAVELAND FLOYD, M.D.*

THE production of artificial pneumothorax is not uncommonly attended with great difficulty on account of the presence of pleural adhesions. These are encountered in many cases because of the fact that pulmonary tuberculosis develops from an initial lesion at the periphery of the lung. Where the pleura is directly involved and an effusion follows, many pleural strands appear and often an adhesive pleuritis occurs with partial obliteration of the pleural space. Many cases of phthisis, that could be benefited by artificial pneumothorax, are abandoned after futile efforts to find a free space. The anti-adhesion pneumothorax needle is designed to meet this need.



The calibre is that of the pneumothorax needles now in use.

Its advantages are, that the blunt stylet can be carried about three-eighths of an inch beyond the end of the needle after it has entered the chest cavity and this allows for separation of the visceral and parietal surfaces of the pleura. The double lock for the stylet at its base permits its being held securely in this position. With the stylet in place, the calibre of the needle barrel allows the passage of air through the needle and into the chest cavity.

The separation of the pleural surfaces not only brings about a high percentage of successful cases, but the opportunity of forcing air into the lung and producing an air embolus is greatly diminished and thus the safety of the procedure is measurably increased.

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THE ELLA SACHS PLOTZ FOUNDATION FOR THE ADVANCEMENT OF SCIENCE

During the twelfth year of the above designated Foundation, seventy applicants for grants were received; thirty-four of this number came from the United States. Of the twenty-five grants made during this year, thirteen were to scientific men in the United States. In the twelve years of the existence of this foundation, it has distributed two hundred and fifty-two grants.

Of the number who have been aided in research work this year, Dr. William Dameshek of Boston received recognition for his work on blood pigment metabolism in lead poisoning. Dr. Charles Lund of Boston for studies on the hormone intermedin and Dr. Yellapragada Subbarow of the Harvard University Medical School for his work on the isolation of materials.

The Thorndike Memorial Laboratory, Boston City Hospital, of which Professor George R. Minot is the Director, has had a continuing grant since 1927 in recognition of Dr. Francis W. Peabody's service to the Foundation.

Applications for grants for the year 1936-1937 must be in the hands of the Executive Committee before May 1, 1936. They should be sent to Dr. Joseph C. Aub, Huntington Memorial Hospital, 695 Huntington Avenue, Boston.

PHYSICAL EXAMINATIONS FOR CITY EMPLOYEES

The Municipal Civil Service Commission of New York City is considering the advisability of providing annual physical examinations for the 90,000 city employees.

The purpose of the plan is to raise the efficiency of the large number of people employed, many of whom are neglectful of health or disinclined to spend the required amount incident to a doctor's fees. Together with the yearly examination, the advisability of including free treatment is under consideration.

The arguments in favor of this plan are based on the experience of industrial organization where it has been shown that care of the health of the workers has so improved the efficiency of the service that money has been saved. Other large cities may be interested in observing this experiment.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 22161

PRESENTATION OF CASE

A fifty-five year old white single woman was admitted complaining of jaundice.

The patient had been perfectly well until three years before entry. At this time she had two attacks of very severe sharp right upper quadrant pain which radiated through to the back and was associated with nausea, vomiting, and very slight jaundice. The duration of each attack was about ten days and both attacks subsided following the local application of ice. She continued well until one year prior to admission when she noted gradual loss of weight. Six months later she had three or four attacks of slight jaundice associated with chills, fever, vertigo, and mild pain in the epigastrium and right upper quadrant. Six weeks ago jaundice appeared and within a week it had become quite intense. A week later unbearable itching developed. She consulted her local physician who prescribed medication and a dietary régime under which her weight dropped quite rapidly. With this attack of jaundice there was no nausea or vomiting but the patient had considerable gaseous eructation and slight epigastric discomfort. The stools became clay-colored, and she developed constipation. The urine became darker in color. Increasing readiness of fatigue was attributed by the patient to dietary restriction. Her total loss in weight during the preceding year was 30 pounds. Generally she did not feel too badly.

The patient had had typhoid fever thirty-eight years before entry. The menopause occurred three years ago.

Physical examination showed a middle-aged woman who was quite deeply jaundiced, rather poorly nourished, in no great discomfort. The sclerae and conjunctivae were markedly icteric and the head and neck were otherwise negative. The heart sounds were of good quality. There was a soft systolic murmur at the apex. The blood pressure was 128/70. The lungs were clear. The abdomen exhibited normal respiratory excursions. There was no tenderness or rigidity. There were no masses palpable. The liver, spleen, and kidneys were not palpable. Rectal and pelvic examinations were negative. The extremities were negative.

The temperature, pulse and respirations were normal.

Examination of the urine was negative. The blood showed a red cell count of 3,830,000, with a hemoglobin of 75 per cent. The white blood cell count was 7,200. The nonprotein nitrogen of the blood was 25 milligrams per cent. A Kahn test was negative. The icteric index was 100. The serum bilirubin was 4.9 milligrams per cent. The stools were definitely clay-colored and tests for occult blood were negative.

A flat plate of the gallbladder region revealed no stones. A gastrointestinal series showed a smooth notch on the greater curvature of the antrum which was probably due to adhesions. There was a large pressure defect on the lesser curvature which was thought to be probably due to gallbladder pressure. The remainder of the duodenum was normal.

In view of the persistent jaundice, operation was decided upon. The patient withstood the operation fairly well, but postoperatively her appetite and strength did not return so quickly as would be expected and she seemed to be slowly getting weaker in spite of the fact that her jaundice was clearing somewhat. After the second week she lost ground noticeably and died about the third week after operation.

DIFFERENTIAL DIAGNOSIS

DR. TRACY B. MALLORY: My role in the presentation of this case is somewhat of a novelty and requires a word of explanation. It was selected from the records of the City Hospital so that I could not possibly know the postmortem findings and I have contracted to attempt the clinical side instead of the pathologic. I am not doing this with any idea of setting an example of how it should be done but just to show that I am not afraid to "take the rap."

We have here a woman of fifty-five with an apparently obvious history of biliary colic on two occasions followed by a period of good health and then the onset of a final illness which lasted about a year. It suggested in the story, and if it is correct I think it is very important, that she began to lose weight nearly six months before the jaundice appeared in this final illness. The jaundice this time is evidently different from that of the two previous occasions. This time she had no pain and instead of being a mild transient jaundice it is a rapidly developing progressive affair. The stools were clay-colored and the urine was dark, apparently. The stools I should assume were uniformly clay-colored; that is, she was getting no bile through at all. So that we have a strong presumption that the jaundice is obstructive.

The physical examination hardly helps us at all. It confirms the jaundice, and the surprising thing is that nothing could be felt in the abdomen, not even the liver, spleen or kidneys.

The laboratory examinations tell us that she had an anemia, apparently of the secondary type, that she did not have any leukopenia, and they confirm the jaundice and the lack of bile pigment in the stools. It seems to me that essentially we have the differential diagnosis of painless jaundice.

Can this be cirrhosis? The fact that all bile pigment is absent from the stools seems to me rather strongly against that diagnosis. We have nothing in the way of positive evidence to point to cirrhosis. They did not feel the liver as they would have if it had been hypertrophic biliary cirrhosis. They could not feel the spleen as they reasonably might have if it had been atrophic cirrhosis with portal obstruction. She did not have leukopenia, which again I would expect with atrophic cirrhosis. The anemia, moreover, was not noticed to show a high color index or to be macrocytic. It seems to me we have nothing in favor of cirrhosis as the primary disease.

Now can we explain the picture with a stone? We have every reason to suppose she had a gallbladder that contained stones. One might have slipped down into the common duct. So far as the jaundice and the absence of bile in the stools are concerned there is no inconsistency. It is perfectly possible that she might not have pain with it, although unlikely. If she had a stone completely obstructing the common duct I would rather expect a big liver, that the bile would have been dammed up behind the stone and would have led to so-called hydrohepatosis, dilatation of the bile ducts and secondary swelling of the liver. That does not have to occur however. I have the impression that when obstruction in the biliary tract develops rapidly enough you sometimes do not get distention of the liver just as with ligation of the ureters you do not get hydronephrosis; but the absence of liver enlargement nevertheless seems to me a point slightly against stone. I do not see how you can rule out stone with any certainty in this case and for that reason I think the surgeon would be not only justified in operating on the patient but obligated to do so. On the other hand I think he would stand a very slim chance of finding a stone in the common duct.

One other common lesion to consider would be malignancy and it seems to me that is strongly suggested by the history of thirty pounds loss in weight which, I think we can trust the history, started before the jaundice appeared. If it is malignancy we have to try to decide where it is located. I do not believe it is primary cancer in the liver because hepatoma occurs in this country only in patients with cirrhosis and, since I have already pointed out that we have no evidence of cirrhosis, I certainly cannot make that diagnosis. It might be a primary cancer of the intrahepatic bile ducts. It would have to be pretty strategically placed, right at the hilum, to catch both the hepatic ducts and cause complete obstruction without being big enough to enlarge the liver. When we consider the last paragraph we find that the surgeon did something which was followed by a temporary relief in the jaundice and with cancer that far up it would have been practically impossible to do anything that could have relieved it. It might be lower down in the bile ducts, in the common duct, or actually at the papilla. I do not think there is any way of ruling out that possibility. It could, of course, be in the head of the pancreas and the laws of probability would be all in our favor if we made such a diagnosis. I rather judge though that considering that she is at the present time described as poorly nourished they might have felt an epigastric tumor if it was cancer of the head of the pancreas. But if it is in the right place it does not have to be very large, so we cannot rule that out.

So far everything has run apparently smoothly enough until we come to the x-ray examination of the stomach and there my troubles begin. They describe what seem to be two different lesions. One is in the greater curvature and is described as a notch. A notch might mean that it was something like a crater but they did not quite dare say it was a crater. It may mean only a localized irregularity. Would that be your interpretation, Dr. Holmes?

DR. GEORGE W. HOLMES: Yes.

DR. TRACY B. MALLORY: On the other side of the stomach, the lesser curvature, there is a large deformity which they interpret as pressure from a mass outside the stomach. An interpretation of that sort could perfectly well be at fault, and it seems to me that one must consider the possibility that we are dealing here with a primary cancer of the stomach but there seems to be very little to go with it. The gastrointestinal symptoms are very slight, the stools show no occult blood, and there was no vomiting in the last illness which one would expect with a cancer as low down in the stomach as the antrum. I am more inclined to accept the x-ray man's interpretation and say that was pressure from outside.

First, we have, presumably, a rather large tumor mass pressing on the lesser curvature of the stomach and then we have something else deforming the greater curvature. I find it pretty hard to conceive of any single mass that could do both these things. It sounds to me more like two masses, and I would be tempted to believe that there is a mass in or on the under surface of the liver which is causing this deformity of the lesser curvature, and that there

is something else perhaps in the region of the pancreas which is involving the greater curvature. These two masses might well be a cancer and its metastases. So again we come to the question of where the cancer is primary. It seems to me it is pure guesswork but I would like to place a first bet on its being primary in the gallbladder with metastases to the nodes at the head of the pancreas, the favorite place to which these cancers metastasize. Cancers of the gallbladder very often invade the liver directly without causing very much enlargement, which would also fit our story. We have to go back at this point, however, and consider the fact that the surgeon operated and did something which temporarily relieved the jaundice. If the gallbladder had been filled with cancer it hardly seems possible that by draining the gallbladder either externally or into the stomach that he could have relieved the jaundice. It is conceivable however that the viscus which was drained was not the gallbladder but was a dilated common duct which could have been anastomosed to the stomach or to the duodenum. In this case we must assume that it was not the primary cancer but its metastases which produced the obstruction.

I have tried to rule out cirrhosis as the primary disease and feel pretty confident about that. Whether there is any secondary liver pathology ought, I think, to be considered at least. There are a certain number of cases of obstructive jaundice associated with stones or malignancy in which one sees the development of an atrophy, a severe toxic necrosis of the liver. It is not inconceivable that she may have had a stone and that operation failed to cure her because she developed a secondary liver insufficiency. That, however, seems unlikely to me because cases of that sort in our experience here have almost always died very promptly after operation. Whether the necrosis in such cases is present before operation is hard to determine, but these cases die within two or three days as a rule rather than slowly petering out two or three weeks later. Another and more likely possibility is a mild biliary cirrhosis of the obstructive type, almost universal in cases of obstructive jaundice. I am prepared to pin all my faith on cancer somewhere in the biliary tract and my first bet in location is the gallbladder.

I would be very glad to hear some opinions from the clinicians.

Dr. Chester M. Jones: I agree with you that they are not going to find gallstones and cirrhosis. I think there is one other condition which is pretty rare, an obstructive type of inflammatory process involving the duct, and tissues around the duct too, which may result in jaundice. I do not know how it could be diagnosed except by exploration. We have had one or two cases here. It would be very hard to see how any surgical treatment except possibly dilatation of the duct could give any relief. Dr Vincent, have you any suggestions?

Dr. Beth Vincent: None, except that obliterative cholangitis, which Dr. Jones says occurs, would not account for the defect in the stomach.

Dr. Jones: It reads very curiously here at the end of the x-ray report. It says "the remainder of the duodenum was normal". I wonder if the terminology was mixed up. Were they talking about the lower end of the duodenum? Having described nothing, they say the remainder is normal.

Dr. Tracy B. Mallory: I could not tell from the report just what that meant and was forced to disregard it.

A Physician: Just assuming the surgeons did help, it is the only thing to bet on. I should say the obstruction is low in the common duct.

Dr. William D. Smith: As far as it goes, my first guess is that it is primary in the gallbladder.

Dr. Tracy B. Mallory: Someone to fall with me. I guess we are ready for the axe to fall.

Clinical Diagnosis

Carcinoma of the head of the pancreas?

Dr. Tracy B. Mallory's Diagnoses

Carcinoma of the biliary tract, probably primary in the gallbladder.
Cholelithiasis.

Anatomic Diagnoses

Papilloma of the papilla of Vater.
Biliary sinus (post-operative) communicating with the common bile duct.
Chronic cholecystitis with cholelithiasis and impacted stones in the cystic duct.
Obstructive cirrhosis.
Icterus.
Pulmonary emphysema.

Pathologic Discussion

Dr. Kenneth Mallory*: There seem to be only two difficulties with your pathologist's clinical diagnoses. The first was the location of the tumor in this case. On inspecting the duodenum we found that the papilla of Vater was enlarged to about one centimeter in diameter. On opening it up we found a papillary mass arising in the ampulla, covering half the surface, and obstructing the ampulla pretty completely, although we could get a probe through it. We thought grossly it would turn out to be carcinoma, instead of which it proved to be a rapidly growing papilloma with no infiltration at the base at all. In addition there was dilatation of the bile ducts above the obstruction measuring one centimeter in diameter.

*Assistant Pathologist, Boston City Hospital.

There was a hydrops of the gallbladder. It was filled with mucoid material, stones and stones impacted in the cystic duct. Secondary to this there was a microscopic grade of biliary, obstructive cirrhosis. We found proliferation of the bile ducts and slight increase in the surrounding fibrous tissue. Also there were obstruction and dilatation of the pancreatic ducts and some proliferation of the finer branches of the pancreatic duct. That is about all.

I think the x-ray findings are rather misleading, because they lead one away from the diagnosis rather than help. I would have brought down the films but I was unable to find them.

A PHYSICIAN: What did the surgeon do? Choledochostomy?

DR. KENNETH MALLORY: That would be my interpretation of the postmortem findings. At autopsy we found a sinus tract which barely admitted a probe that led down into the common bile duct just above the ampulla.

I think this papilloma of the ampulla of Vater is rather rare. We have had carcinomas of course. Dr. Castleman showed me one case from your records here at the Massachusetts General which is also papilloma rather than cancer of the ampulla.

CASE 22162

PRESENTATION OF CASE

A twenty-seven year old American laborer was admitted complaining of shortness of breath.

Five months before entry the patient, who had been perfectly well previously, suddenly coughed up a clot of blood about the size of a half dollar. A physician examined him and found nothing of significance. A specimen of sputum was examined and found to be negative. A week later, following strenuous exertion, he coughed up small quantities of bright red blood for about one hour. Another examination showed nothing, but an x-ray done at this time was said to be negative for tuberculosis although some other process in the chest was noted. Subsequently the patient developed a persistent cough, usually brought on by exertion, which was productive of a small amount of blood-tinged sputum. At scattered intervals he complained of dull pain in the left chest. Gradually dyspnea with exertion developed and occasionally he had wheezing respirations. Three months before entry he suffered a chill and a sharp pain in the left chest, which was aggravated by respiratory movements. A physician made a diagnosis of pneumonia and he remained in a hospital for two weeks. During his acute illness he coughed up quantities of yellowish sputum, which was occasionally streaked with blood. He developed frequent night sweats which persisted up to his admission. Following his recovery from the "pneumonia", dyspnea with moderate exertion became quite marked and he began to prop himself with pillows at night in order to sleep more comfortably. The cough continued and became dry and brassy in character.

Physical examination showed a well-developed and nourished young man sitting propped up in bed with slight respiratory difficulty. The lips and fingernails were cyanotic. There was no generalized adenopathy, but the epitrochlear nodes were palpable. The heart appeared to be displaced to the right and the apex impulse was in the fourth interspace, 6.5 centimeters from the midsternal line. The right border of cardiac dullness was 4.5 centimeters from the midsternal line. The heart sounds were normal. The examiner thought that the trachea was slightly deviated to the left. Chest expansion was limited on the left side. The left chest was dull to percussion anteriorly and posteriorly, and flat posteriorly beneath the angle of the scapula. Grocco's triangle was elicited although the side was not noted. Breath sounds and tactile fremitus were diminished to absent from above downward in the left chest. A few musical râles were audible bilaterally, more on the left side.

The temperature was 99°, the pulse 80. The respirations were 20.

Examination of the urine was negative. The blood showed a red cell count of 5,300,000, with a hemoglobin of 80 per cent. The white cell count was 17,100, 78 per cent polymorphonuclears. Several sputum examinations were negative for tubercle bacilli. A Hinton test was negative.

X-ray examination showed the right side of the diaphragm and the right lung to be negative. The posterior two-thirds of the left leaf of the diaphragm was obscured. It was said to be definitely elevated at fluoroscopy. There was a triangular area of density with the shape of the lower lobe, but about one-third as large, occupying the posterior and inferior portions of the left chest. Just above this triangle at the level of the lung root and slightly posterior to it was a rounded mass, 3 centimeters in diameter. This shadow contained no air. The heart and mediastinum were displaced toward the left. Several films taken at intervals previously during the patient's illness showed very little change in the size of the mass since the initial film but there was a progressive collapse of the left lower lobe and at one time the entire left lung was collapsed. Later films taken at expiration and inspiration showed ball valve occlusion of the upper lobe bronchus.

On the sixth day the patient had a chill and his temperature rose to 102°. The throat was found to be inflamed. This subsided promptly and two days later a bronchoscopy was done. This showed a firm, whitish, roughly pyramidal

tumor mass about one inch below the carina in the left main bronchus. On the following day the patient had a temperature of 101.6° and coughed a great deal. Increased dullness was elicited in the left upper chest and bronchial breathing was audible in the upper chest posteriorly. No râles were present. The temperature rose to 103.2° on the following day but subsequently returned to normal and the patient remained comparatively comfortable. Following three administrations of pneumothorax, on the twenty-third hospital day a left thoracotomy was performed.

Differential Diagnosis

Dr. Frederick T. Lord: The history of hemoptysis out of a clear sky is especially suggestive of pulmonary tuberculosis or of tumor. With the continuing decline in the number of cases of tuberculosis, tumor becomes relatively more common as a cause. Other possible causes of hemoptysis out of a clear sky are syphilitic ulceration of the trachea and bronchi, bronchiectasis and the rupture of tuberculous glands into the air passages. Echinococcus disease and Distoma ringeri would have to be considered as exotic causes. The wheezing suggests bronchial obstruction and it would be desirable to know its relation to posture and to cough and expectoration.

Assuming the presence of bronchial obstruction, the acute illness three months ago with chill, pain, cough and blood-streaked sputum may be ascribed to pneumonia arising in consequence of the trapping of infection behind an obstruction.

The brassy cough is difficult of explanation. It suggests the possibility of tracheal obstruction.

A biopsy on an enlarged gland may establish the diagnosis, but epitrochlear glands do not seem a likely site of metastasis.

The apparent displacement of the heart to the right is confusing and it would be desirable to know the site of maximum intensity of the heart sounds. It is unusual to have the heart displaced to the right and the trachea to the left.

Grocco's triangle of paravertebral dullness on the unaffected side is seldom of diagnostic value and least helpful in the presence of small pleural effusions where we most need assistance.

The signs in the left chest, dullness to flatness and diminished to absent breath sounds and tactile fremitus, though consistent with a small pleural effusion, are suggestive under all circumstances of a closed bronchus and consequent atelectasis.

Dr. Tracy B. Mallory: We have no x-ray man here but I will ask Dr. Churchill to show the films.

Dr. Edward D. Churchill: The first x-ray was taken September 3, 1935. Knowing what this abnormal area at the left hilum is, it is not fair to give the interpretation. This is a film of ours taken in February at the time of his admission. This is an expiration film taken the same day. There is quite a difference between the two films on full inspiration and full expiration.

Dr. Lord: On comparison of the film at the end of full inspiration with that at the end of full expiration, it is evident that air enters the right side more readily than the left. On the left side in the region of the left border of the heart is a shadow with an unusually straight and sharply limited margin. This extends upward to the region of the left lung root. In the film during inspiration with the heart displaced to the right, the sharply limited shadow persists and suggests the presence of a collapsed left lower lobe.

Dr. Churchill: The swing in the mediastinum is rather interesting and may account somewhat for the confusing physical signs.

This is an x-ray taken after artificial pneumothorax.

Dr. Lord: There is a small pneumothorax in the lower and a larger amount of air in the upper part of the left side, with an intervening area of lung extending to the chest wall. The appearance suggests air above and below a portion of the lung adherent to the chest wall.

Dr. Churchill: The x-ray interpretation suggested that in addition to the collapse of the lower lobe there was a trapping of air in the upper lobe. The upper lobe did not deflate with expiration as much as it should have.

Dr. Lord: There is some asymmetry in the films. The trachea is about in the middle line and it is difficult to say whether it is displaced.

Dr. Churchill: Dr. Hampton's interpretation was an obstructing lesion presumably represented by this opaque area at the left hilum which has not changed in size since September, and which he thought now had caused complete obstruction of the lower lobe bronchus with collapse of the lower lobe and probable partial ball valve obstruction of the upper lobe without collapse.

Dr. Lord: Ball valve action seems more common with foreign body than with tumor, but at some time in the course of gradual encroachment on the bronchial lumen by an expanding tumor, inspiratory widening and expiratory narrowing of the passage may be expected to lead to the more ready entrance than exit of air and consequent pulmonary inflation.

It would be desirable, if possible, to know from the x-ray examination of any broadening of the carina. I cannot make out the bifurcation of the trachea on these films and it would be difficult to tell without the use of lipiodol. It would be desirable to have x-rays

of such other regions as the skull, spine and long bones. Was that done?

DR. CHURCHILL: No.

DR. LORD: The presence of metastases would materially change the problem. Also, by bronchoscopy it would be desirable to determine the mobility of the trachea and bronchi.

In explanation of the bronchial breathing in the upper left chest behind, it may be due to a pneumonic process or to the persistence of atelectasis after the release of bronchial obstruction.

The statement that a tumor in the left primary bronchus has been established by bronchoscopy narrows the discussion. In addition, there is atelectasis of the left lower lobe. There is likely also to be some bronchopneumonia of the left lower lobe, and there may also be bronchiectasis and abscess formation in the same region. By x-ray he had obstructive emphysema of the left upper lobe.

It is of importance to remember with respect to the site of the tumor that, although it presents itself by bronchoscopy one inch below the bifurcation of the trachea, the origin of the tumor may arise from more remote bronchi and project upward into the lumen of the primary bronchus without attachment to the bronchial wall. There may thus be more space between the carina and the site of origin of the tumor than appears from the bronchoscopic description.

With respect to the nature of the tumor, inflammatory narrowing is the most common cause of bronchial obstruction. Granulation tissue may, however, be dismissed as unlikely here. The chances are in favor of carcinoma. It may, however, be benign adenoma, a fibroma or polyp, but I make the diagnosis of a malignant tumor of bronchial origin and that implies carcinoma. The next question is naturally with respect to metastases, and x-rays of other parts of the body would help. Metastases into the brain occur in about a quarter of such cases and into the adrenal in about a third, and may be scattered elsewhere. Months or even years may elapse without metastases and there is evidence from the investigation of Tuttle and Womack (*J. Thoracic Surg.*, Dec. 1934) that malignant tumors of the major bronchi extend more slowly and are thus more amenable to surgery than those arising in the minor bronchi.

It is perhaps premature to discuss treatment, but I am tempted to do so in view of the statement that a thoracotomy was performed. As it is spoken of as thoracotomy without qualification, it was very likely an exploratory operation and by this means metastases may have been seen or felt in the pleural space and their presence or absence probably determined the question of further surgical procedure.

There is nothing but surgery which offers any considerable promise of success. X-ray treatment is not successful with deep-seated cancer, but may give temporary relief with lymphoblastoma. There are rare instances as in the case reported by Jackson and one by Arbuckle, in which bronchoscopic removal has been successful. Implantation of radium may be used in cases not amenable to surgery, but there is little to recommend the method and there is danger of a reaction from the radium beyond the region to which it is applied. Thus with malignancy, surgery is the only promising expedient. Naturally in so difficult a problem as bronchial malignancy the operative mortality is high, but otherwise there is only the prospect of a fatality.

DR. CHURCHILL: The surgeon's hand was strengthened in this case by bronchoscopy, which gave a positive diagnosis of very rapidly growing carcinoma; this, despite the fact that it is of five months' duration in a healthy appearing young man of twenty-seven. The one disturbing feature about the case surgically was the observation by bronchoscopy that the growth extended one inch from the carina in the main stem bronchus. Dr. Lord has spoken of a tendency for cancers of the lung to grow from a tumor into a bronchus without invading the mucosa of the bronchial wall. It seemed quite possible that we were facing that situation. If there had been definite evidence of invasion of the bronchial wall within an inch from the carina, operation would not have been advised. A lantern slide will demonstrate an autopsy specimen from a patient that gave us this concept. This patient came to autopsy several months after pneumonectomy, having died of a local recurrence at the site of amputation of the bronchus and you see the thumb of tissue extending up the left main bronchus, passing the carina and extending into the trachea but showing no attachment to the bronchial mucosa until we come down to the main body of the tumor. So in approaching the operation on the patient we are now discussing, our problem was to anticipate just such a situation. Consequently, instead of putting a clamp or ligature around the left primary bronchus it was carefully divided with the knife and the "thumb" of tumor extracted from the bronchus.

This slide shows the lung when resected with the plug of tumor projecting from the left main bronchus. This is a closer view. This sloughing ulcerating tumor had been described by the bronchoscopist as pyramidal in shape. This is the dissection of the lung and you can see the pus pouring out of the lower lobe as the plug of tumor was lifted upward relieving the obstruction.

The man died of infection. Due to manipulation of the infected lung and the ulcerating necrotic tumor in the bronchus his pleural cavity became infected. On the third day it was drained as an empyema. In the presence of in-

fection the bronchial suture gave way so that an open communication between the free pleural cavity and the bronchus developed. A friction rub appeared over the precordium and he died on about the seventh or eighth day of fulminating sepsis, empyema, and mediastinitis. Physiologically he withstood the removal of the left lung. Infection, however, decided the issue.

Clinical Diagnoses

Carcinoma of the lung.
Empyema.

Dr. Frederick T. Lord's Diagnoses

Bronchiogenic carcinoma.
Obstruction atelectasis.
Obstruction emphysema.
Bronchopneumonia?
Bronchiectasis?
Abscesses?

Anatomic Diagnoses

Carcinoma of the lung.
Operative wound: left pneumonectomy.
Empyema, left.
Pericarditis, acute serofibrinous.
Bronchopneumonia, confluent, right lower and middle lobes.
Pulmonary edema, right.
Pleuritis, chronic fibrous, right apical; acute fibrinous, left.

Pathologic Discussion

Dr. Mallory: I will pass around the specimen of resected lung. It shows very prettily a tumor mass, which is of considerable size, out in the parenchyma of the lung and the plug of tumor growing up the bronchus. The tumor apparently actually arose in a small bronchus near the apex of the lower lobe. It grew up the bronchus of the lower lobe and then up the primary bronchus, passing across and partially obstructing in a ball valve fashion, as the clinical evidence shows, the bronchus to the upper lobe. So that it fulfills very completely the predictions of the x-ray department on that score. We have made sections of all of the hilus glands and they were completely free from tumor. We also made a number of sections of the wall of the primary bronchus and were unable to find invasion up to the point of section. There was one point where there appeared to be a slight attachment of the tumor "thumb" to one of the smaller carinas and it had begun to pick up a new nourishment from that point but in the major bronchi it was entirely free.

A Physician: What cell-type is it?

Dr. Mallory: That is not easy to say. It is an unusually undifferentiated tumor composed of very small, very uniform, rather round cells. They seem to be without question epithelial. I do not believe we are dealing with a lymphoma for a moment but it is not a typical epidermoid and it is not like the so-called oat cell tumor. The bronchus at one spot did show squamous cell metaplasia—a lesion which is possibly precancerous. In any case it is very surprising that with such a rapidly growing tumor the regional lymph nodes were free and we found no metastases elsewhere in the body.

The autopsy added comparatively little information. There was of course empyema and rather diffuse bronchopneumonia in the lung on the opposite side. There was some infection of the mediastinum and a septic pericarditis. Those are all the usual complications that would be feared in a case of this sort. We found no evidence of metastases although we had no permission to do the head. It is possible there may have been cerebral metastases.

Dr. Churchill: About the bronchoscopic biopsies, I have removed either one or more lobes or the entire lung in sixteen cases of tumor. In only nine of that number were there positive bronchoscopic biopsies. The impression given in a number of articles now is that bronchoscopy is the certain way to diagnose carcinoma of the lung. It is a certain way, but if you wait for positive bronchoscopic biopsies, you are going to miss a great many cancers of the lung at a time when it may be possible to do something for them surgically.

Of the sixteen cases five are living and apparently well; two patients received real palliative relief although they died subsequently of their disease; and seven died in the hospital subsequent to the operation. One patient died of widespread metastases, having experienced no real palliative relief and one patient with a benign tumor has been greatly relieved but will require additional surgery. In nine of this series of patients a total pneumonectomy has been performed. Four of these survived the operation and were discharged from the hospital; five died in the hospital.

The New England
Journal of Medicine

SUCCESSOR TO
THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

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SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 Fenway.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The New England Journal of Medicine, 8 Fenway, Boston, Mass.

THE CAUSE OF CORONARY DISEASE

THE long arm of coincidence is responsible for much medical folly—scientific as well as empiric. An alleged increase in the frequency of appendicitis has been ascribed to various causes, from the steel milling of wheat flour to the swallowing of raspberry pips. Many are the vagaries with which the unknown may be illuminated by an imaginative soul, yet where in the evolution of intellect is a single concept or achievement devoid of imagination? Imagination is the fire of thought; it warms an otherwise barren outlook; it illuminates otherwise invisible possibilities. But imagination, like fire, cannot be safely allowed out of control; when it destroys facts it must be stamped out; when it threatens reason it must be all but extinguished. Coincidence is the first check to apply to any imaginative conception; it is the primitive correlation.

No subject is of more present speculative interest among medical doctors than that of coronary disease. All but the youngest of us have had our friends and contemporaries struck down by it. All but the oldest of us are willing to follow any rational program of prevention—or to advise a rational program for others if it seems too arduous for personal practice. There is no lack of coincidences to lead us on, but life is too sweet to follow them all, and they are apt to contradict one another, too.

It is perhaps only a coincidence that three Boston doctors have suggested within the past year, that we might profitably steer in one general direction. Their observations have not been correlated studies, but it would seem that they have unintentionally converged on the same general point from three different angles.

Dr. Timothy Leary has rather conclusively established coronary sclerosis as part of a more generalized metabolic disease, akin to diabetes, and associated with cholesterol deposits in the subendothelial layers of the arteries.[1] He has, moreover, produced this type of "atherosclerosis" in rabbits by cholesterol feeding. He suggests that our coronary vessels may be exposed to these changes by two circumstances: 1. The inheritance of a weak cholesterol metabolism. 2. The overdosage with cholesterol containing food in the dietary.

Dr. Cadis Phipps has more recently published an analysis of contributory causes of coronary thrombosis.[2] He also mentions the possible influence of overeating, not from the point of view of cholesterol dosage, but as a general precipitating cause. In addition, Phipps doubts that physical stress is a great factor, indeed he suggests a better future for the manual laborer with heart disease than for the white collar worker.

In this issue of the *Journal* (page 769), Dr. Francis P. Denny presents a statistical review of the death certificates for heart disease in Brookline, Massachusetts, since 1900. One sees therein an evolution of medical thought from the days when every murmur meant endocarditis, through the increasing recognition of "arteriosclerotic" and "hypertensive" heart disease, to the present popularity of "coronary disease". Back of it all, however, is the unequivocal increase in death from coronary disease. Denny's foremost speculation is that insufficient or irregular muscular activity is the cause of this increase.

One might almost suspect that these three men had frequently lunched together in order to correlate their views before expressing them. Their views—based upon separate primitive coincidences—themselves coincide to give us a fairly direct hygiene for the prevention of coronary disease. The initial step in instituting this, as in all preventive hygiene, is the choice of good heredity. Within the limits thus imposed we may further advise and practice dietary discretion, both qualitatively and quantitatively, as well as regularity of physical exercise. This latter should adjust itself to the

oxidation of the foodstuff eaten—particularly the fatty foodstuff eaten. Such hygiene might grow in wisdom with the years, and if it worked—ah, if it worked—what a relief it would be to hear less often that heart disease is the leading cause of death!

REFERENCES

1. Leary, Timothy: Atherosclerosis, important form of arteriosclerosis, metabolic disease; eleventh Ludvig Hektoen lecture of Frank Billings Foundation of Institute of Medicine of Chicago. J. A. M. A. **105**: 475 (Aug. 17) 1935.
2. Phipps, Cadis: Contributory causes of coronary thrombosis. J. A. M. A. **106**: 761 (March 7) 1936.

THE PROBLEM OF SILICOSIS

Newspaper reports of the development of silicosis in large numbers of rock drillers in a West Virginia tunnel have recently called attention to the extent of this hazard in certain occupations which involve exposure to silica dust. In New York City it is estimated that at least 4,000 people, notably men engaged in the excavation of foundations and subway tunnels, are exposed to silica and other dust hazards, and the city Health Department is now studying means to reduce this exposure. Last winter the New York Legislature passed a workmen's compensation act, including silicosis among compensable occupational diseases.

Lanza and Vane have estimated that in the United States alone not less than 500,000 workers are, through their occupations, subject to a harmful exposure to silica dust. Metal mining, quarrying, stone-cutting and the foundry industry are the greatest sources of exposure, but potteries, glass works and such special processes as sand blasting, grinding, polishing and hard-rock drilling in coal mines contribute very materially to the production of silicosis. The losses from silicosis in terms of sickness and death are even more difficult to estimate, but it can be said with little fear of contradiction that silicosis now constitutes the greatest single occupational hazard in this country.

The causal relationship of constant exposure to rock dust to such conditions as "miner's phthisis" and "stonecutter's consumption" has been recognized for centuries but only recently has the distinction between silicosis and tuberculosis been generally accepted or the coincidence of the two diseases understood. Chiefly as a result of the investigations of members of the South African Institute for Medical Research and of Gardner at Saranac Lake the theory of mechanical injury from the inhalation of stone dust has been replaced by evidence of chemical action. Briefly, the minute silica particles retained in the air passages are ingested by wandering cells which in turn carry them to adjacent lymph nodes. In the body fluids, however, the silica is slightly soluble and toxic to the phagocytic cells. Many of the dust-laden cells are killed in this way and their load is picked up by other cells. The silica also stimulates the fibroblasts with which it comes in contact, resulting in a scarring, first of the lymph nodes and perivascular structures, and finally in overgrowth and replacement of the respiratory epithelium.

Under working conditions the development of silicosis is dependent upon a number of variables, which can be measured only by highly technical procedures. Among the most important of these factors are percentage of free silica in the dust inhaled; number of particles per unit of air; size of particles inhaled; duration of exposure to silica dust; and susceptibility of the individual exposed. Experience has shown that massive exposure to dust high in silica, as in the manufacture of abrasive cleaning compounds, may produce disabling silicosis in a year or even less, while thirty-five years in a granite shed leaves a few cutters without even x-ray signs of pulmonary disease. Yet in most industries it is possible to estimate the hazard of silicosis with a degree of accuracy unusual in medical problems.

Uncomplicated silicosis is the most insidious of diseases. A slight dyspnea after perhaps fifteen or twenty years of exposure to dust is likely to be the first warning to the worker. Even then the condition tends to be slowly progressive and may not be disabling during the span of working life. The fibrosis, however, is an irreversible reaction and a process once established may progress to a fatal termination as long as fifteen or twenty years after exposure to silica has ceased. The real hazard from silicosis is the tuberculous infection which so frequently accompanies it and which is a menace to both the worker and his associates. Various studies indicate that from 25 to 50 per cent of silicoties also have tuberculosis and it is estimated that 75 per cent of all patients with silicosis die of tuberculosis.

That silicosis is not an abstract problem in Massachusetts was forcibly brought home to us two years ago by the report of the Special Industrial Disease Commission appointed by the General Court. X-ray and physical examination of representative groups of granite and foundry workers disclosed silicosis in 15 per cent and silicosis complicated by tuberculosis in 7.6 per cent of the former, compared with 8.8 per cent and 2.6 per cent respectively in the latter, and this represents the incidence among men actually at work. A study of mortality among granite cutters in two cities for a twelve-year period showed a tuberculosis death rate ten times that among all males of twenty years and over in the same cities.

Although Massachusetts was one of the first states to declare silicosis a compensable disease, a worker discharged on account of a diagnosis of silicosis still has to prove physical disabil-

ity to entitle him to compensation, and his success is likely to depend on the medical and legal counsel he is able to employ. Compensation insurance rates for granite workers have risen to a level that has caused most Massachusetts employers to withdraw from their employees the protection of the Workmen's Compensation Act, which is not compulsory in this state. The unfortunate situation thus created applies to granite workers either disabled as a result of previous dust exposure or injured through industrial accident. The carefully worked out plan of the Industrial Disease Commission which aimed to reduce dustiness to safe levels, provide compulsory insurance coverage for hazardous occupations, with compensation for disabled and tuberculous workers and to detect pulmonary disease by periodic physical examinations at the hands of impartial examiners, was agreed upon in principle by employers, employees and insurance companies, yet no group was willing to make the concession necessary to put it into effect. Until labor and industry can make common cause against a mutual loss, medical science can do little toward the prevention of this greatest occupational disease.

REFERENCES

1. Lanza, A. J., and Vane, R. J.: Am. Rev. Tuberc. **29:** 8 (Jan.) 1934.
2. Gye, W. E., and Purdy, W. J.: Brit. J. Exper. Path. **3:** 86 (April) 1922.
3. Mavrogordato, A.: Pub. of So. African Inst. for Med. Research. No. 15 (March 31) 1922.
4. Gardner, L. U., and Cummings, D. E.: Am. J. Path. (supp.) **9:** 751, 1933.
5. Russell, A. E., et al.: Public Health Bulletin No. 187, Washington, 1929.
6. Report of Special Indust. Dis. Commission, Commonwealth of Mass. House No. 1350 (Feb.) 1934.

CLEANLINESS NEXT TO GODLINESS

CLEANLINESS among school children, and the need for health instruction in cleanliness, is the thesis of Rowell and Tobey, publishing their views on the subject in the *American Journal of Public Health* (**25:** 1237, [November] 1935). Godliness is not mentioned, even by inference. Various inadequacies in facilities for cleanliness now exist, to greater or less degree, in the majority of American schools. Thus, according to these authors, in 1928 it was found from an investigation of 404 schools located in 22 states and the Dominion of Canada, that in only 53.2 per cent of the buildings was hot water supplied, in 80 per cent was some form of soap furnished, and in 84.1 per cent were towels available.

Only 32 per cent of these schools, however, could comply with the American Child Health Association's standards of one lavatory to every 80 pupils, equipped with all three of the necessities—hot water, soap of sorts and towels, only 19.3 per cent complied with the requirements of the Massachusetts Institute of Technology, specifying one lavatory to every 40 pupils, and only 5.7 per cent conformed to the Wood-Rowell standards of at least one lavatory, equipped with hot water, soap and towels, and conveniently located, to every 20 pupils.

Obviously, the lesson of cleanliness once learned cannot be practiced by the pupil and will not be carried home to a benighted family unless a reasonable opportunity is afforded of pursuing the laboratory course in the subject. The authors, however, go beyond an insistence on the mere motions of ablution. The quality of the soap should be within certain standards of excellence, and should be adapted to the chemical nature of the water available, in which regard the judgment of the average school janitor, excellent as it might be in other respects, should not be considered as necessarily of expert calibre. Soaps should be selected, then, with a view to their germicidal properties, their action on the skin, and their reaction with the water, the general belief being that the purest soaps, made from high quality fat and possessed of a relatively low content of free alkali, are the most germicidal.

The incidence of infectious disease has been reduced by many public health measures; still greater reduction, it is hoped, can be attained by the widespread practice of personal cleanliness.

SUCCESS

WHAT is success and who may attain it? Is the surgeon successful? Yes, though his case may have a fatal outcome; for he, thoroughly trained in the intricacies of his technic, has given of his best.

And what of the pathologist who labors industriously, painstakingly and unceasingly to discover that elusive germ, the deadly enemy of mankind, that causes some dread disease which one day will be but a bitter memory? Surely he is successful, though many hours of patient effort may seem a total loss. How often when he is most discouraged does he find that the solution has been almost within his grasp! He cannot and will not allow discouragement to turn him from his task, but will rather resolve to persevere that his goal may be attained.

Then too, there is the medical missionary, that remarkable contributor to medical service in Labrador, Dr. Grenfell for example, who, with few resources and many handicaps, performs by saving the child whose life has been despaired of what seems to the anxious parents little less than a miracle. How often he is also rewarded by the lifelong gratitude of the man whose suffering has been almost intolerable, or the woman who has gone through "the valley of the shadow of death".

And, of course, there is the general practitioner. His efforts are never-ceasing, and though called upon to do much that demands unlimited

self-sacrifice he seldom receives an adequate recompense. But that does not deter him. He continues through many years of patient effort, and becomes the friend of all who know him.

May we not, then, say with truth that all whose efforts are honest, painstaking and productive of the best they have to offer, are successful? Not only physicians but nurses, technicians, and innumerable others may with propriety be included among those who justly claim success. Is it not the duty of all of us to co-operate in our daily tasks, performing them to the best of our ability and remembering that, though our success may seem very meager indeed, the result of our labors must of necessity help toward the advancement of that great science Medicine, which will continue in its development throughout all time.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MCGINN, SYLVESTER. A.B., M.D. Harvard University Medical School 1929. Assistant in Medicine, Massachusetts General Hospital, and St. Elizabeth's Hospital. Consultant, Sturdy Memorial Hospital. Address: 270 Commonwealth Avenue, Boston, Mass. Associated with him is

WHITE, PAUL D. A.B., M.D. Harvard University Medical School 1911. Physician, Massachusetts General Hospital. Assistant Professor of Medicine, Harvard University Medical School. Address: Massachusetts General Hospital, Boston, Mass. Their subject is Progress in the Recognition of Congenital Heart Disease. Page 763.

DENNY, FRANCIS P. A.B., M.D. Harvard University Medical School 1895. Health Officer, Board of Health, Brookline, Mass. His subject is The Increase in Coronary Disease and Its Cause. Page 769. Address: 111 High Street, Brookline, Mass.

TENNEY, BENJAMIN, JR. B.S., M.D. Harvard University Medical School 1925. F.A.C.S. Assistant Obstetrician, Massachusetts General Hospital and Cambridge Hospital. Junior Visiting Surgeon in Obstetrics and Gynecology, Boston City Hospital. Assistant in Obstetrics, Harvard University Medical School. His subject is A Clinical and Pathological Study of One Hundred and Fifty Cases of Tubal Pregnancy. Page 773. Address: 309 Marlboro Street, Boston, Mass.

DARRAH, L. W. M.D. University of Pittsburgh School of Medicine 1911. Assistant Physician, Gardner State Hospital. His subject is Congenital Absence of the Vermiform Appendix in a Patient with Mental Disease. Page 776. Address: East Gardner, Mass.

YOUNG, ALBERT G. B.S., Ph.D., M.D. Harvard University Medical School 1928. Formerly, Research Pharmacologist, University of Wisconsin; Assistant Professor of Pharmacology, University of Michigan; Assistant in Medicine, Boston City Hospital; Research Fellow in Medicine, Thorndike Memorial Laboratory; and Member of Medical Staff, Boston Dispensary. Now, Assistant Professor of Therapeutics, Boston University School of Medicine, and Member of Staff, Evans Memorial Hospital. His subject is The Occurrence of Allergic Reactions in Arthritic Patients. Page 779. Address: 520 Commonwealth Avenue, Boston, Mass.

GRAVES, ROGER C. A.B., M.D. Syracuse University College of Medicine 1918. F.A.C.S. Urologist, Carney Hospital. Genito-Urinary Surgeon, Pondville Hospital. Member of the Associate Staff, New England Deaconess and Palmer Memorial Hospitals. Consulting Urologist, Quincy City Hospital, Lakeville State Sanatorium and Winchester Hospital. Genito-Urinary Consultant, Tumor Clinic, Boston Dispensary. Associate Consulting Urologist, Brockton Hospital. Address: 12 Bay State Road, Boston, Mass. Associated with him is

KICKHAM, C. J. E. A.B., M.D. Harvard University Medical School 1927. Assistant Urologist, Carney Hospital, Assistant Visiting Urologist, Pondville Cancer Hospital at Norfolk. Associate Consulting Urologist, Quincy Hospital. Address: 12 Bay State Road, Boston, Mass. Their subject is Congo Red for the Control of Bleeding. Page 782.

PETERSON, THOMAS H. A.B., M.D. Northwestern University Medical School 1926. F.A.C.S. Instructor in Orthopedic Surgery, Harvard University Medical School. Junior Visiting Surgeon, Boston City Hospital. Assistant Visiting Orthopedic Surgeon, Long Island Hospital. Associate Orthopedic Surgeon, New England Baptist Hospital. His subject is Method of Applying A Temporary Adhesive Support to the Back. Page 783. Address: 23 Bay State Road, Boston, Mass.

FLOYD, CLEAVELAND. M.D. Harvard University Medical School 1903. Physician-in-Chief, Division of Tuberculosis, Boston Health Department. His subject is A New Instrument. An Anti-Adhesion Pneumothorax Needle. Page 785. Address: 246 Marlboro Street, Boston, Mass.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY*

C. J. KICKHAM, M.D., *Chairman*
524 Commonwealth Ave., Boston, Mass.

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472 Commonwealth Ave., Boston, Mass.

POSTPARTUM HEMORRHAGE

PART 2

If the uterus is carefully and continuously held after the birth of the baby, one knows definitely whether it is changing in size. If it is not changing in size and if there is no descent of the cord and no bleeding, one knows definitely that the placenta is still attached. If this condition continues for one-half to three-quarters of an hour, one begins to suspect the presence of an adherent placenta. If there has been descent of the cord and subsequent bleeding, one suspects that the placenta is separated and possibly retained. Now, of course, all adherent placentas are retained but not all retained placentas are adherent. The completely adherent placenta shows no evidence of bleeding. The retained placenta will show some evidence of bleeding and occasionally unusual bleeding, but if the fundus has been continuously held and is not overlarge, one knows that there is no concealed hemorrhage. I think there should be no worry, as long as there is no real hemorrhage, concerning a placenta that has not been delivered within an hour of the birth of the baby, and of course if there is no hemorrhage the length of time that the placenta remains in utero is not important, up to a certain point. However, if there have been evidences of separation or if there is some bleeding and the placenta has not been delivered within one hour or one hour and a half, it is very wise to investigate the uterus. This, of course, must be done under most careful asepsis, as entering the uterus after delivery is attended by a real risk of infection.

It is wise to have the patient anesthetized. One hand is put into the uterus, the other hand gives intelligent suprafundal pressure. If the placenta is a retained one and lying free in the uterine cavity behind a contracted os, the cervix is dilated and the entire placenta readily removed. It is rare that a case of this sort will bleed subsequently. It is consequently rare that in a case of this sort the uterus needs to be packed.

The adherent placenta offers a very different problem. If it is completely adherent there will have been no hemorrhage, no bleeding. If it is partially adherent and partially separated there may have been a tremendous amount of bleeding. In either case the hand is thrust into the uterus and if one finds a partially separated placenta one readily recognizes the line of cleavage. If possible, the entire placenta should be removed in one maneuver. Those cases in which the placenta is partially separated may have bled and may bleed after the delivery of the placenta—so much so that packing the uterus is oftentimes a wise procedure. If the uterus has been packed the tamponade should be left in not over twenty-four hours, and the fundus should be held so that one is certain that there is no bleeding behind the pack. If the adherent placenta is entirely adherent, one attempts to find the line of cleavage. This is usually possible but it is often impossible to remove all the placenta in one maneuver, and these cases more often require packing than any other class.

Now there is one other type of retained placenta and that is the placenta accreta. The placenta accreta, if it is a complete placenta accreta, will give no bleeding at all. Upon investigation some line of cleavage is found but one soon appreciates that this line of cleavage stops. One realizes that the placenta is embedded in the uterine wall—a part of the uterus. As soon as this diagnosis has been made, the proper procedure is abdominal section and hysterectomy. There is one type of accreta very infrequently seen in which the placenta practically covers the entire uterine wall. The thickness of the placenta may be practically no more than one-quarter inch. This, too, when the diagnosis is made demands hysterectomy.

Now it matters not whether a patient bleeds from a lacerated cervix or from the rupture of varicose veins or from a partially separated, adherent placenta or from an accreta. It does not matter what the cause of postpartum hemorrhage is. Loss of blood itself demands treatment. Postpartum hemorrhage very frequently requires transfusion and it is a very wise practice in any case of this sort to have an acceptable donor at hand before any operative procedures are instituted. Subsequent transfusions may be necessary; also intravenous glucose, clysis, the repeated use of intramuscular ergot and pituitrin and—par excellence—the use of morphine; but once the source of the bleeding is diagnosed and controlled the treatment is the treatment of hemorrhage. It matters not whether this comes from a perforated gastric ulcer, a bleeding tonsil artery or a lacerated cervix. The treatment of postpartum hemorrhage, when the cause is diagnosed and the bleeding stopped, is the treatment of hemorrhage, and that often means actual blood transfusion.

*A series of short selected articles by members of the Section is being published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

AIDS TO THE COMMITTEE OF ARRANGEMENTS

NORFOLK DISTRICT

Dr. J. S. H. Leard.
Dr. Herbert L. Johnson.
Dr. H. M. Landesman.

A PRIZE FOR AN APPROVED ESSAY

The attention of interns in Massachusetts hospitals is called to the fact that a prize of $50.00 has been offered by the Massachusetts Medical Society for the best written and most comprehensive case report submitted by one of their number holding a rotating internship in any Massachusetts hospital which is approved by the American Medical Association for intern training during 1935-1936.

This report is to be typewritten, and when completed is to be sealed, unsigned, in a plain envelope, which in turn is to be placed together with a separate slip bearing the name and address of the contestant, in a larger envelope, and sent to:

The Massachusetts Medical Society,
Committee on Medical Education and Medical Diplomas
8 Fenway, Boston, Mass.

The contest this year closes May 1, 1936. Reports may be submitted at any time prior to that date.

THIRD ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The following sessions have been arranged by the Committee for the week beginning April 19:

Berkshire

Thursday, April 23, at 4:30 P.M., at the House of Mercy Hospital, Pittsfield. Subject: Kidney and Bladder Diseases A (Medical)—Acute Nephritis — Etiology, Diagnosis and Treatment. Nephrosis and Its Treatment. Instructor: W. R. Ohler. Melvin H. Walker, Jr., Chairman.

Bristol North

Wednesday, April 22, at 7:30 P.M., at the Morton Hospital, Taunton. Subject: Lung Diseases (Surgical)—(a) Empyema. (b) The Value of Surgery in Chronic Lung Diseases; Tuberculosis, Lung Abscess, etc. Instructor: R. H. Swett. Arthur R. Crandell, Chairman.

Bristol South (New Bedford Section)

Friday, April 24, at 4 P.M., at the St. Luke's Hospital, New Bedford. Subject: Dermatology—Ten Common Skin Diseases—Diagnosis and Treatment. (1) Impetigo Contagiosa, (2) Scabies, (3) Acne Vulgaris, (4) Psoriasis and Seborrhoeic Dermatitis, (5) Epidermophytosis, (6) Herpes Simplex and Zoster, (7) Eczema, (8) Erythema Multiforme, (9) Verruca Vulgaris and (10) Dermatitis Medicamentosa and Dermatitis Venenata. Instructor: John Adams, Jr. Harold E. Perry, Chairman.

Franklin

Wednesday, April 22, at 8:00 P.M., at the Franklin County Public Hospital, Greenfield. Subject: Cancer of the Stomach and Bowel. Instructor: L. S. McKittrick. Halbert G. Stetson, Chairman.

Middlesex East

Wednesday, April 22, at 4:00 P.M., at the Melrose Hospital, Melrose. Subject: Kidney and Bladder Diseases A (Medical)—Acute Nephritis — Etiology, Diagnosis and Treatment. Nephrosis and Its Treatment. Instructor: L. H. Hoyt. Joseph H. Fay, Chairman.

Middlesex North

Friday, April 24, at 7:00 P.M., at the Lowell General Hospital, Lowell. Subject: Arthritis. Instructor: F. R. Ober. Leonard C. Dursthoff, Chairman.

Norfolk

Friday, April 24, at 8:30 P.M., at the Norwood Hospital, Norwood. Subject: Lung Diseases—(a) Differential Diagnosis and Treatment of Lobar Pneumonia. (b) The Surgical Problems of Empyema. Instructors: J. H. Pratt and J. W. Strieder. H. B. C. Riemer, Chairman.

Worcester (Milford Section)

Wednesday, April 22, at 8:30 P.M., at the Milford Hospital, Milford. Subject: Lung Diseases—(a) Differential Diagnosis and Treatment of Lobar Pneumonia. (b) The Surgical Problems of Empyema. Instructors: H. F. Newton and D. S. King. Joseph I. Ashkins, Sub-Chairman.

MISCELLANY

CONNECTICUT ITEMS

TRI-CITY MEDICAL SOCIETY OF NORWICH, NEW LONDON, AND WILLIMANTIC, CONNECTICUT

The regular monthly meeting of the Tri-City Medical Society of Norwich, New London, and Willimantic, Connecticut, was held Thursday, March 12, 1936, at Uncas-On-Thames, Norwich. Dr. Richard Cattell of the Lahey Clinic, Boston, delivered an address on The Management of Surgical Diseases of the Colon and Rectum.

The principal manifestations of *ulcerative colitis* are diarrhea, fever, toxemia and an elevated white blood count. In the usual case, proctoscopic examination means establishing the diagnosis. These cases are poor surgical risks. Dr. Cattell believes that any patient with an acute fulminating colitis who does not show early improvement should be operated upon. The disease is a medical condition first but if no relief is obtained medically, surgery should be resorted to. However, the operative mortality is high. Medical treatment is often very inadequate because of the anatomical changes caused by scarring due to loss of mucosa. The etiology of the disease has not been proved, but it probably is

a nonspecific infection which usually begins in the rectum. Ulceration may perforate and cause peritonitis. At the Lahey Clinic these cases are usually operated upon in several stages. A transverse ileostomy is done, followed in the second and third stages by removal of the colon. In a number of cases, complete colectomies have been done with a cure of the condition. In 100 cases receiving early attention, partial colectomy has eradicated the disease. However, in most cases complete colectomy is necessary.

In considering *differential diagnosis* of colon lesions it is necessary to use digital examination, proctoscopic examination, and x-ray of the colon. At the clinic, a double contrast air and barium enema has been found helpful.

The *treatment* of *diverticulitis* is essentially medical. In a few instances surgery becomes necessary. When this is the case, it must be done in two stages, the first stage consisting in a colostomy or cecostomy above the lesion.

Polyps, Dr. Cattell says, are the main etiological factor in carcinoma or malignancy of the large bowel and so any polyp discovered should be adequately treated. Usually these polyps can be cured by fulguration through the sigmoidoscope.

At the Lahey Clinic, where 500 cases of cancer of the rectum and colon have been carefully analyzed, it is felt that there is a good chance of a cure if properly treated. Early diagnosis is essential for good results. The best working rule is to do the most radical operation for the lesion that the patient will stand.

The New London County Medical Association

At a meeting of this organization, April 2, the following officers were elected for the ensuing year: Dr. William T. Driscoll of Norwich, Conn., President; Dr. Thomas Soltz of New London, Conn., Vice-President; Dr. George H. Gildersleeve of Norwich, Secretary-Treasurer; Dr. Charles G. Barnum of Groton, Conn., Councilor; Censors, Drs. Daniel Sullivan, New London; James J. Donohue, Norwich, and David Sussler, Norwich. State Delegates elected were Drs. George H. Gildersleeve and Albert C. Freeman of Norwich and Drs. Richard Starr and Isidore Hendel of New London.

After the business meeting Dr. Charles F. Wilinsky of Boston gave a most excellent informal talk on The Economic Aspects of Medicine. A general discussion followed.

The meeting ended with a supper served by the Sanatorium.

Death

CHILDS—Helen Simonds Childs, M.D., aged eighty-four, died at Simsbury, Connecticut, April 8, 1936, at the home of her daughter, Mrs. John Schroeder, after a brief illness. A son also survives her.

Dr. Childs formerly practiced in Jamaica Plain, Massachusetts.

CHANGES IN THE FACULTY OF BOSTON UNIVERSITY SCHOOL OF MEDICINE

Dr. John Arthur Foley has been promoted from the position of Associate Professor of Medicine to Clinical Professor of Medicine, and Dr. William Reid Morrison from the position of Assistant Professor of Surgery to Clinical Professor of Surgery.

FELLOWSHIPS AT THE HARVARD MEDICAL SCHOOL

Ten of the major fellowships at the Harvard Medical School, totaling $10,150, have been awarded for the next academic year, as follows:

William O. Moseley, Jr., Traveling Fellowship, for the study of medicine in Europe, to Champ Lyons, assistant in bacteriology, Harvard Medical School, Harvard M.D. 1931.

William O. Moseley, Jr., Traveling Fellowship, for the study of medicine in Europe, to Louis H. Nason, M.D. '31, of Boston, Mass.

Edward Hickling Bradford Fellowship, for medical research, to Alwin M. Pappenheimer, Jr., Ph.D. '32, has been at National Institute for Medical Research, Hampstead, London, England.

William Hunter Workman Fellowship, for postgraduate study in this country or abroad, to Theodore H. Ingalls, M.D. '33, New Hartford, Conn.

Jeffrey Richardson Fellowship, for postgraduate study in this country or abroad, to Benjamin V. White, Jr., M.D. '34, Summit, N. J.

James Jackson Cabot Fellowship, for medical research, to John H. Dingle, 1M., Kalamazoo, Mich.

John Ware Memorial Fellowship, for medical research, to Richard L. Riley, 3M., Plainfield, N. J.

Charles Eliot Ware Memorial Fellowship, for medical research, to Emanual B. Schoenbach, 3M., New York City.

George Cheyne Shattuck Memorial Fellowship, for medical research, to Israel Kapnick, 2M., Providence, R. I.

DeLamar Student Research Fellowship, to Charles B. Burbank, 2M., Miami, Fla.

AN ASSIGNMENT TO ATTEND THE MEETING OF THE MASSACHUSETTS MEDICAL SOCIETY

Senior Surgeon Louis Schwartz. Directed to proceed from New York, N. Y., on or about June 8, 1936, to Springfield, Mass., and return, to attend the meeting of the Massachusetts Medical Society, on June 8, 1936, and to present a paper on the subject of Industrial Dermatoses.

THE HOLYOKE BOARD OF HEALTH

A new board of health of the city of Holyoke has been appointed by the Mayor, consisting of Dr. Edwin M. Mahoney, Dr. Joseph A. Wonsik, and Mr. Arthur Hebert.

The board organized with Mr. Hebert, chairman, and Dr. Mahoney, secretary.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1935 AND SEVEN YEAR AVERAGE

MONTH ENDING MARCH 28, 1936

Diseases	1936 Week ending Mar. 7	1936 Week ending Mar. 14	1936 Week ending Mar. 21	1936 Week ending Mar. 28	Average cases reported for week corresponding to Mar. 28 for past seven years	1935 Week ending Mar. 9	1935 Week ending Mar. 16	1935 Week ending Mar. 23	1935 Week ending Mar. 30
Chickenpox	116	113	106	80	82	123	177	130	98
Conjunctivitis Infectious	3	6	7	5	2	—	—	3	1
Diphtheria	2	2	3	4	13	7	—	8	7
Encephalitis Epidemic	—	1	2	—	—	—	—	3	—
German Measles	248	329	257	649	35	191	145	180	131
Influenza	26	25	48	9	22	2	9	4	28
Measles	89	88	85	79	297	997	878	1213	1448
Meningococcus Meningitis	1	2	1	3	1	3	—	—	—
Mumps	65	67	78	46	107	60	70	151	81
Paratyphoid Fever	—	—	—	—	—	—	1	—	1
Pneumonia (Broncho)	49	51	61	31	38	27	56	45	51
Pneumonia (Lobar)	100	76	88	61	54	62	41	49	59
Poliomyelitis	—	1	—	—	—	—	—	—	1
Scarlet Fever	126	150	113	102	120	70	95	121	116
Streptococcus Sore Throat	6	3	7	3	3	3	1	1	6
Tetanus	—	—	—	—	—	1	—	—	—
Trachoma	—	—	—	1	—	—	—	—	—
Trichinosis	—	—	3	—	—	1	—	—	1
Tuberculosis (Pul.)	19	22	17	33	32	35	30	15	19
Tuberculosis (O. F.)	—	4	3	2	4	—	2	1	1
Typhoid Fever	—	1	1	1	—	—	—	—	3
Undulant Fever	2	3	—	2	—	2	—	—	—
Whooping Cough	99	110	86	117	75	97	64	45	100
Gonorrhea	22	20	12	22	27	39	19	17	38
Syphilis	54	57	45	47	42	49	33	34	42

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

A FRENCH TRIBUTE TO RESEARCH

The President of the French Republic has been pleased to confer upon Sir Henry Wellcome, LL.D., F.R.S., la Croix d'Officier de la Légion d'Honneur.

This decoration is a further tribute to medical and chemical research to which Sir Henry has made many notable contributions.

MARTYRS TO SCIENCE

Forty American radiologists were named in Hamburg, Germany, April 4, among the 165 recorded as heroes of science who sacrificed their lives in medical service.

In the list the name of Walter J. Dodd of Boston appears. The names are chiseled on a simple sandstone memorial in front of the Roentgen Institute of St. George's Hospital.

The names of forty-six French and seventeen German scientists are included.

LORD HORDER WILL FILL THE POSITION OF PHYSICIAN-IN-CHIEF, PRO TEMPORE, AT THE PETER BENT BRIGHAM HOSPITAL

On the first of May of this year, Lord Horder, Physician-In-Ordinary to the King and Senior Physician at St. Bartholomew's Hospital, London, will be at the Peter Bent Brigham Hospital as Physician-in-Chief pro tempore.

TUBERCULOSIS IN NEW YORK CITY

Dr. John L. Rice, Health Commissioner of New York City, is quoted in the daily paper as reporting a marked increase in tuberculosis in the city.

The number reported for the first quarter of 1936 for New York City is 2,955 as contrasted with 2,568 last year and 2,785 in 1934. The mortality due to tuberculosis has also risen slightly. The increased incidence of tuberculosis is ascribed to the effect of the depression.

DR. PARRAN HAS BEEN SWORN IN

Dr. Thomas Parran, Jr., was sworn in, April 6, as Surgeon General of the United States to fill the position made vacant by the resignation of Dr. Hugh S. Cumming.

CORRESPONDENCE

THE INSPECTION AND REPORT OF FLOODED AREAS IN MASSACHUSETTS

The Commonwealth of Massachusetts
Department of Public Health
State House, Boston

April 7, 1936.

Managing Editor, *The New England Journal of Medicine*.

A request was made to the United States Public Health Service by His Excellency, the Governor, asking that a sanitary engineer be sent to make an inspection of the flooded areas and report on the conditions found with reference to health and sanitation. Dr. C. C. Applewhite and Sanitary Engineer Arthur P. Miller were assigned to this work. After an inspection of the Merrimack and Connecticut Valleys, their report, a copy of which is enclosed, was sent to the Surgeon General.

HENRY D. CHADWICK, M.D.,
Commissioner of Public Health.

COPY OF THE REPORT

March 26, 1936.

The Surgeon General,
U. S. Public Health Service,
Washington, D. C.
Sir:

In compliance with telephonic orders received on March 23, we proceeded immediately to the State of Massachusetts for duty in the flooded areas. Upon arrival in Boston, we reported to Dr. Chadwick, State Health Commissioner, and at his request, we made a survey of the Merrimack River Valley with him on March 24. On that date, the Merrimack had receded for the most part from the business and residential sections and the work of rehabilitation was progressing satisfactorily in practically all of the areas.

In this survey we confined our attention to those factors which would have a definite bearing on the spread of diseases. The most serious situation encountered by us in the Merrimack Valley was found in Lawrence. Here, the water filtration plant which is located on the banks of the river and the water pumping station which is adjacent to the filters had been entirely flooded. Fortunately, this city's reserve supply of purified water was adequate to maintain a continuity in distribution until arrangements could be made to increase the quantity in storage by borrowing water from three neighboring communities. However, as work proceeded in rehabilitating the water purification plant, it was soon found necessary to make some curtailment in the uses of water in the municipality. These were brought about, however, without seriously inconveniencing the public. With these temporary measures in force, exploratory work at the filtration plant continued as rapidly as possible and by March 25, two groups of slow-sand filters had been unwatered and it was possible to put decreased quantities through them into the storage reservoir. Every precaution was taken during this period by the State Department of Health through its Division of Engineering to insure that water distributed to the public was satisfactory as to bacteriological quality. The situation at Lawrence can be summed up by saying that water of safe quality is now available to the city, although in slightly curtailed quantities.

At Lowell, Massachusetts, the water purification plant and the pumping station were both put out of commission by flood waters. Fortunately, the physical layout of this system was such that the raw water sources, which are driven wells near the bank of the river, were not seriously affected and an adequate supply was constantly available. Precautions were taken, however, to disinfect this water at two points on the system, one in the conduit carrying the water to the city and the other at the pumping station located in the city proper. Through these means, a continuous supply of pure water was furnished to Lowell at all times and no evidence was available to indicate that any outbreak of waterborne diseases would be possible.

In both Lawrence and Lowell, the prompt and adequate measures, which were put into force by the State Department of Public Health, entailing men on duty 24 hours a day, assured continuous supplies of water suitable for public use.

After a fairly complete survey of the Merrimack Valley towns affected by the flood, it was felt that all measures essential to the protection of the public health were being put into execution in a satisfactory manner.

At the request of Dr. Chadwick, we proceeded to the flooded area in the Connecticut Valley on March 25. The following towns were visited in company with a member of the staff of the State Department of Public Health, Springfield, Holyoke, Northampton, Hatfield, Deerfield, Chicopee.

Upon our arrival in Springfield, it was learned that all of the public water supplies in the Connecticut Valley which might have been adversely affected had already been adequately safeguarded as a result of the alertness and vigilance of the District Engineer in that territory. In this area, a survey was made of the facilities for caring for those evacuated from the flood area and the measures which were being put into execution for the purpose of reclaiming the flooded area.

Most of the towns had adequate physical facilities for taking care of those who were removed from the flooded area in school houses and other public buildings. A detailed inspection of a school thus utilized at Springfield was made. At this school, about 800 people were being housed and fed. The sanitary conditions at the time of inspection were beyond criticism, and the precautions taken for protecting

the people against the dangers incident to overcrowding were considered adequate, and in all the towns visited there was no report of an increase in the number of communicable diseases.

The sections of all municipalities, which had been flooded, were protected by the National Guard and police to prevent looting. Just as rapidly as the water receded, inspections of the homes were being made by the police and Health Departments to determine the safety of the dwellings for occupancy. The task of ridding the dwellings, stores, streets, and sidewalks of mud, slime, and débris deposited by the flood is large and time-consuming. However, this material which will have to be removed in order to make the places fit for human habitation is of no consequence so far as disease causation is concerned. In most of the towns, considerable progress has already been made in this clean-up campaign.

Having in mind the emergency conditions, which obtained in each of these stricken valleys, both of us were of the opinion that satisfactory provisions had been made to guard against the spread of communicable diseases and that those engaged in efforts toward this end should be commended for promptly initiating and executing the measures essential to safeguarding the public health.

C. C. APPLEWHITE, *Surgeon*,
ARTHUR P. MILLER, *Sanitary Engineer*.

RECENT DEATHS

McEVOY—THOMAS EDWARD MCEVOY, M.D., of 769 Main Street, Worcester, Massachusetts, died in that city, March 28, 1936.

Dr. McEvoy was born in 1859 and educated in the public schools of Hopkinton, Massachusetts, Exeter Academy and Yale. He graduated from the Yale Medical School in 1892, served his interneship in the Bridgeport City Hospital, later being associated with the teaching staff of the Lying-In Hospital of New York City.

Soon after settling in Worcester in 1896, he was appointed a member of the staff of St. Vincent's Hospital and served in that capacity until about five years ago when he became consultant.

His widow, Mrs. Mary W. (Spencer) O'Day-McEvoy; a son, Thomas S. McEvoy, and a stepdaughter, Miss Marion T. O'Day, survive him.

CURTIS—FRANCIS GEORGE CURTIS, M.D., of Ashfield, Massachusetts, died at his summer home there, April 7, 1936.

Dr. Curtis was born in Staten Island, New York, in 1857, graduated from Harvard College in 1879 and from the College of Physicians and Surgeons of New York in 1883. He had practiced in Newton, Mass., until twenty years ago and had been the Chairman of the Newton Board of Health for forty-two years, resigning this position last July because of ill health.

Dr. Curtis joined the Massachusetts Medical Society in 1887 and was also a Fellow of the American Medical Association, a member of the Newton Medical Club, the Staff of the Newton Hospital and one of the founders of the Massachusetts Association of Boards of Health and had served as president of this last-named organization.

He is survived by his widow, Mrs. Ruth Curtis, and three sons, Mr. George Curtis, Mr. Shaw Curtis, and Mr. Edward Curtis.

DAVENPORT—FRANCIS HENRY DAVENPORT, M.D., a retired physician of Boston, died April 9, 1936, at the Hotel Puritan. Dr. Davenport was born in Roxbury in 1851, graduated from Williams College and later received his M.D. degree from the Harvard Medical School in 1874. After postgraduate courses in Vienna and other medical centers, he returned to Boston and was an instructor in gynecology at Harvard with an affiliation at the Massachusetts General Hospital. Dr. Davenport joined the Massachusetts Medical Society in 1877 and retired in 1935.

He was a member of the Harvard and the Williams Clubs and the Gynecological Society of America.

A son, Henry Davenport, survives him. His wife died in 1905.

NORTON—EBEN CARVER NORTON, M.D., of North Chatham, Massachusetts, died at his home, April 11, 1936, after an extended illness. Dr. Norton was born in 1856 at Vinal Haven, Maine, the son of Jesse and Hannah Carver Norton. He was a graduate of Harvard College and of the University of Vermont Medical School.

Dr. Norton was credited as the founder of the Norwood Hospital for mentally ill patients. His previous experience in psychiatry was at the State Hospital at Tewksbury and in association with Dr. Walter Channing.

He moved to North Chatham in 1928. He was a Fellow of the Massachusetts Medical Society and the American Medical Association.

A son, E. Lawrence Norton, of Salt Lake City, Utah, and three daughters, Miss Helen Norton of Chatham, Mrs. Howard E. Whipple of Richmond, Virginia, and Mrs. Gregg Smith of Long Beach, California, survive him.

KNIGHT—CHARLES STORER KNIGHT, M.D., died in Portland, Maine, April 12, 1936, following a recent operation in Boston.

Dr. Knight was born in Portland, Maine, and after his preliminary education in Portland schools and Westbrook Seminary, entered the Harvard Medical School and graduated in 1896.

He was a former house physician at the Boston City and the Boston Lying-in Hospitals and at one time was assistant superintendent of the Boston City Hospital. He was a Fellow of the Massachusetts Medical Society from 1896 to 1905.

NOTICES

THE HENRY JACKSON LECTURES OFFERED BY THE NEW ENGLAND HEART ASSOCIATION

These lectures will be given by Tinsley R. Harrison, M.D., Associate Professor of Medicine, Vanderbilt University School of Medicine, at 4:45 P.M. on Thursday, April 30, and Friday, May 1, at the Boston Medical Library (John Ware Hall).

Subjects:

1. The Pathogenesis of Circulatory Failure.
2. The Principles of Therapy in Patients with Congestive Heart Failure.

Physicians and students of medicine are cordially invited to attend.

The annual business meeting of the New England Heart Association will precede the lecture on April 30.

ANNUAL TUFTS ALUMNI ADDRESS

Dr. William B. Keeler, Class of 1903, Health Commissioner for the City of Boston, will deliver the annual alumni address to the students of Tufts College Medical School, Wednesday, April 29, at four o'clock in the amphitheater at the school, 416 Huntington Avenue. The subject of his address will be "Preventive Medicine and Public Health." The alumni, faculty, and friends of the school are cordially invited to attend.

MEDICAL CLINIC AND STAFF ROUNDS AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, April 23, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian.

THE GEORGE WASHINGTON GAY LECTURE

Dr. W. Dacre Walker, a graduate of Tufts College Medical School in 1905, will give the George Washington Gay Lecture on Wednesday, April 22, at four o'clock in the amphitheatre at Tufts College Medical School. Dr. Walker has chosen as his subject: "What the Small Town Doctor Does."

The George Washington Gay Lectureship Fund was originally established in 1926 with a gift from Dr. George W. Gay of Chestnut Hill, Massachusetts for lectures at the Tufts College Medical School upon Medical Ethics and the Art and Practice of Medicine.

FIRST INTERNATIONAL CONGRESS OF SANATORIA AND PRIVATE NURSING HOMES

The First International Congress of Sanatoria and Private Nursing Homes will be held in Budapest at the end of September next. Special invitations will be sent in due course to all parties concerned. Lectures, discussions, proposals, etc., should be sent to the following address as early as possible in order that they may be included in the official program: Committee of the First International Congress of Sanatoria and Private Nursing Homes, Budapest, Margitsziget, Sanatorium.

PHYSICIANS' ART EXHIBITION

The Physicians' Art Society will hold its annual exhibition April 29-May 9 at Doll & Richards Galleries at 138 Newbury Street, Boston.

All members of the Massachusetts Medical Society who do painting and sculpture are cordially invited to join the Society and show examples of their work at the coming exhibition.

Application should be made to James F. Ballard at 8 Fenway, Boston.

CLOVER HILL HOSPITAL
Lawrence, Mass.

The next speaker in the Clover Hill Hospital series of monthly medical lectures will be Donald Munro, M.D., Visiting Surgeon for Neurosurgery to Boston City Hospital. His subject is The General Practitioner's Relation to the Problem of Brain and Skull Injuries.

Date: Thursday, April 23, 1936 at 8:30 P.M. His talk will be illustrated.

All physicians of Greater Lawrence are invited to attend.

NICANDRO F. DECESARE, M.D., *Chairman.*

REPORTS AND NOTICES OF MEETINGS

NATIONAL HEALTH COUNCIL
ANNUAL MEETING

The annual meeting of the National Health Council, held in New York City on February 6, was of unusual interest this year because of the election of new officers and the discussion of a series of problems of significance in national health conservation.

The Nominating Committee reported that it found it necessary to relieve Colonel Roosevelt from the Presidency because of business, professional and national public services, which precluded continuance of his administrative duties in connection with the Council. Expressing regret that such action had become necessary, the Committee recorded its recognition and deep appreciation of the services Colonel Roosevelt had rendered the Council, and its satisfaction in reporting that he would continue as a member at large of the Board of Directors.

The Nominating Committee then recommended the election of Dr. Donald B. Armstrong, as President, for the year 1936, pointing out that he had been an active officer and executive in promoting the Council's development, and had made the final study of all the voluntary agencies which led to its organization in 1920. In addition to Dr. Armstrong's intimate knowledge of all the member agencies and their respective fields of activity, it was recognized that his training and experience in scientific, medical and public health work constituted exceptional qualifications. The Council unanimously approved his selection for the presidency.

For Vice-President, Mr. T. N. Pfeiffer, an attorney, the present incumbent, and widely known for his promotion of social work activities, was nominated.

For Treasurer, Mr. Frederick Osborn, likewise the present incumbent, and a leader in population studies, and other fields of science and welfare, was nominated.

For Secretary, Professor Maurice A. Bigelow, a noted educator and biologist and Professor in Columbia University, was nominated.

These general officers were unanimously elected.

For membership in the Board of Directors ten members at large were nominated, representing public health administration, education and research, as well as eleven direct representatives of the member agencies of the Council. The following is a list of these directors, as unanimously elected:

Dr. R. M. Atwater, American Public Health Association.

Colonel H. E. Bullis, National Committee for Mental Hygiene.

Mr. L. H. Carris, National Society for the Prevention of Blindness.

Miss Dorothy Deming, National Organization for Public Health Nursing.

Mr. Howard Green, National Committee of Health Council Executives.

Dr. Kendall Emerson, National Tuberculosis Association.

Dr. C. C. Little, American Society for the Control of Cancer.

Dr. H. M. Marvin, American Heart Association.

Dr. S. H. Osborn, Conference of State and Provincial Health Authorities of North America.

Dr. William F. Snow, American Social Hygiene Association.

Dr. F. N. Sperry, American Society for the Hard of Hearing.

AT LARGE

Dr. George Baehr, New York Academy of Medicine.

Dr. S. J. Crumbine, Formerly American Child Health Association.

Dr. H. S. Cumming, U. S. Public Health Service.

Dr. William De Kleine, American Red Cross.

Dr. Louis I. Dublin, American Public Health Association.

Livingston Farrand, President, Cornell University.

Mr. Homer Folks, New York State Charities Aid Association.

Dr. A. S. Knight, National Committee for Mental Hygiene.

Colonel Theodore Roosevelt.

Ray Lyman Wilbur, President, Stanford University.

HARVARD MEDICAL SOCIETY

The Harvard Medical Society met at the Peter Bent Brigham Hospital Feb. 11, 1936, Dr. Elliott P. Joslin presiding.

The medical case was presented by Dr. G. L. Sullivan, Jr.: A fifty-three year old, Russian housewife entered the hospital Jan. 17, 1936, complaining of a dry mouth and throat of a few days' duration. Eight years ago she suffered easy fatigue and loss of weight, and was found to have diabetes mellitus, for which she was given insulin and dietary régime. For the past two years she had not used insulin. For the past three weeks she had noticed increasing thirst, and for four days before entry had experienced increasing loss of strength and appetite, with some nausea. On the day of entry there was vomiting.

Physical examination showed a dehydrated, middle-aged woman with an odor of acetone on her breath. There was auricular fibrillation. Examination of the urine showed a trace of albumin, and positive tests for sugar and acetone. Her blood sugar on entry was 433 mg. per cent. The blood urea nitrogen was 21 mg. per cent and the CO_2 combining power 22 vol. per cent. She was placed on the usual diabetic régime, and recovered from her acidosis in satisfactory manner. She had had a continually elevated temperature since admission, which was believed to be due to urinary tract infection, since there were numerous white blood cells found in her urine. X-ray studies of the chest were negative. She required 45 units of insulin before breakfast and 10 units at noon and evening to keep her urine free of sugar. It was decided to try the effect of the new insulin protamine, and she was given 20 units that evening.

Dr. Fitz remarked that she had auricular fibrillation on entry, and that as soon as her acidotic state had been corrected her heart had become regular. He asked if such observations were usual. Dr. Howard F. Root of the Joslin Clinic replied that one similar case had been seen at the Deaconess Hospital.

Dr. Elliott P. Joslin remarked that, of a series of 176 diabetics on whom postmortem studies had been performed, 18 per cent had been found to have active inflammatory disease of the urinary tract. He also stated that insulin protamine required two or three days to exert its full effect.

The second case was presented by Dr. Robert Bates of the surgical service. A twenty-seven year old white housewife entered the hospital with the history of pleurisy on the right side four years ago, since which time she had noted easy fatigue,

general weakness, and diarrhea of severity sufficient to necessitate eight or nine movements each day. Eight months previously her sister died with tuberculosis, and x-ray studies performed on the patient revealed bilateral apical tuberculous lesions. She was hospitalized at Middlesex Sanatorium at that time, where she remained until she was transferred to the Brigham Hospital. One month previous to admission she developed an increasing severity of diarrhea, and x-ray studies revealed marked contraction of the colon. She was transferred to the Brigham Hospital for operation. Physical and laboratory studies were not remarkable. Under spinal anesthesia the terminal ileum, cecum and ascending colon were resected for tuberculous colitis. Her postoperative course was uneventful.

Dr. John Homans stated that this was an ideal case for resection of the colon. He believes that more cases of tuberculous colitis should be operated upon if localization of the disease process can be established.

Dr. Merrill C. Sosman demonstrated the preoperative x-rays of the case, and pointed out that the bowel was not only markedly constricted in diameter, but that it had also greatly decreased in length due to shrinkage, a feature characteristic of tuberculous colitis. The bowel distal to the lesion was shown to be dilated by the barium enema, a feature which Dr. Sosman stated was observed whenever pressure had to be used to force barium through an obstructing lesion, and which was not due to any intrinsic disease in the distended bowel.

The paper of the evening was presented by Dr. C. N. H. Long of the University of Pennsylvania, who spoke on "The Effect of Hypophysectomy and Adrenalectomy upon Experimental Diabetes in the Cat." The removal of all insulin by pancreatectomy gives rise to a series of events:

(1) Hyperglycemia and glycosuria appear, and persist even during fasting.
(2) There is an increase in nitrogen excretion, and there is a definite ratio between the amounts of glucose and nitrogen excreted ("D/N ratio").
(3) Large quantities of acetone bodies appear in the urine.
(4) Finally acidosis develops, coma intervenes, and death occurs.

These facts are established, but the mechanism of the observed events has proved the subject of much controversy. One group holds that the loss of insulin from the body results in an inability of the tissues to utilize carbohydrates, and as a result fats and lipids are drawn upon to furnish body energy. The incomplete oxidation of these substances in the absence of carbohydrate utilization liberates ketone bodies which give rise to the symptoms of acidosis. They believe that there is a definite ratio between the utilization of fat and glucose, the so-called ketogenic-antiketogenic ratio.

The second school of thought is known as the "overproduction theory of diabetes". Their original theory postulated that the removal of the pancreas precipitated a "glandular imbalance" between the thyroid and adrenal medulla, the secretions of the two glands acting in such manner as to cause an overproduction of glucose from fat and protein. This overproduction was so great as to cause hyperglycemia and glycosuria, and was supposed to take place in the liver. It was believed that the tissues maintained their normal ability to utilize carbohydrate, in spite of the absence of insulin. This original theory has not been supported by experimental data, and it has been shown that the body loses its ability to use glucose in the absence of insulin.

Both theories, however, maintain that sugar can be formed from protein by the liver (gluconeogenesis).

A large percentage of individuals dying from diabetes have no demonstrable pathology of the pancreas. Certain patients with abnormalities of other glands are frequently known to show glycosuria, or frank diabetes. Thus glycosuria is observed in some 29 per cent of acromegalics. Cases with increased activity of the adrenal cortex (tumor or hyperplasia) often exhibit glycosuria. These latter cases also exhibit a hyperplasia and hyalinization of the basophilic elements of the anterior pituitary.

The close psychological relationship between the pituitary and adrenal cortex is interesting. Removal of the pituitary causes a shrinkage of the adrenal cortex, with a condensation of the reticular zone, and a decrease in the amount of lipoid material in the zona glomerulata. If anterior pituitary extract is supplied, these changes disappear and the gland returns to normal appearance.

Houssay somewhat elucidated the disturbances in carbohydrate metabolism observed in diseases of the anterior pituitary. Removal of the hypophysis was found to prolong the life of depancreatized animals, and to lower the D/N ratio. Acidosis and ketosis did not appear in these animals, and if fasted they often became sugar-free. (Thus the D/N ratio disappeared.) Injections of crude extracts of the anterior pituitary accentuated the glycosuria, acetonuria, and ketosis.

These depancreatized hypophysectomized animals alternate between hypo- and hyperglycemia. When they are extremely emaciated and their fasting blood sugar is low, administration of intravenous glucose solution is followed by a variation in blood sugar level which is almost identical with a normal blood sugar tolerance curve. If, however, the animals have a high blood sugar level, or are on the usual protein, fat, low carbohydrate diet, administration of glucose results in a typical diabetic blood sugar curve. The R. Q. of these animals increases only very slightly, showing that there is no great increase in the utilization of carbohydrates. Similar results are obtained in depancreatized adrenalectomized animals. It is concluded that neither

hypophysectomy nor adrenalectomy has any pronounced effect in increasing carbohydrate utilization except in emaciated animals with low blood sugar.

There is a marked effect of hypophysectomy on the duration of life and on the fat metabolism of depancreatized animals. The usual life of a depancreatized cat is about five days, and death occurs following extreme hyperglycemia and acetonuria. Depancreatized, hypophysectomized animals have an average life of forty-nine days, their blood sugar is maintained at a lower level and there is a marked decrease in the excretion of glucose, nitrogen and acetone bodies. Thus the chief effect of hypophysectomy seems to be to decrease the production of glucose from protein and the prevention of acidosis. (The marked fall of basal metabolic rate following ablation of the pituitary must not be forgotten, however.)

Removal of the thymus gland had little effect on depancreatized animals. Thyroidectomy, although not increasing the duration of life, or lowering the blood or urine sugar, did decrease acetonuria.

The effect of adrenalectomy on depancreatized animals was studied by Dr. Long and his associates. Removal or denervation of the medulla had no effect in protecting against glycosuria. Removal of both adrenals and the pancreas increased the duration of life, and lowered the excretion of glucose, nitrogen, and acetone bodies. These animals were maintained on large doses of cortical extract. It thus appears that adrenalectomy has an effect similar to that observed after hypophysectomy, although the former animals must be maintained on doses of cortical extract. Hypophysectomized animals do exhibit an atrophy of certain elements of the adrenal cortex, however.

A comparison of the livers of depancreatized animals with or without adrenalectomy shows that there is a greater store of glycogen in the former. The liver fatty acids are usually increased from six to twenty-five per cent following pancreatectomy, an increase which is prevented by removal of the adrenals or hypophyses.

It is important to note that the adrenalectomized animals were maintained on large doses of cortical extract, and that their sodium metabolism was not disturbed. Evidently some cortical principle other than those contained in the extracts used was removed by adrenalectomy. It is possible that the adrenal acts through the pituitary in some way, and that removal of the cortical material produces an effect because of changes in the pituitary secretion.

Injections of potent anterior pituitary extracts were made into depancreatized animals in which the first group had been subjected to hypophysectomy, and the second group to adrenalectomy. The injections into the hypophysectomized animals caused the reappearance of glycosuria, ketonuria and acidosis, but had no effect on the adrenalectomized animals. The specific adrenotropic principle of the pituitary likewise had no effect on the second group of animals, but increased the glucose, but not the acetone body excretion of the first group.

Dr. Long concluded by stating that although the evidence is suggestive, it is by no means conclusive that there is a diabetogenic hormone in the adrenal cortex. Hypophysectomy and adrenalectomy are valuable procedures for the experimental study of diabetes, but they should not be considered as therapeutic measures to be employed in the treatment of the disease.

THE NEW ENGLAND HOSPITAL ASSOCIATION

The New England Hospital Association held its fourteenth meeting February 27, 28 and 29 at the Hotel Statler, Boston. A most interesting program had been arranged.

Mrs. B. B. Marble, Secretary of the American Dietetic Association and Research Dietitian of the Collis P. Huntington Hospital, Boston, presented a very well-edited history of the growth of the national dietetic association. She carefully traced the gradual increase in the importance of the dietitian's functions as the physician's ally in the treatment of diseases and the continuance of investigative work.

Quindara Oliver Dodge, Associate Professor of Institutional Management, Simmons College, Boston, gave a concise discourse on the need and functions of a capable and well-trained manager for the various necessary maintenance services of a hospital.

Thursday afternoon was devoted to the questions which arise in hospitals relative to their training schools. Sister Francis James, St. Mary's Hospital, Waterbury, Conn., presented the educational features available in a well-conducted Out-Patient Department. Miss Elizabeth E. Sullivan, R.N., Supervisor of Schools of Nursing of the Massachusetts State Board of Registration of Nurses, Boston, presented the difficulties which are besetting the various hospitals conducting Training Schools for Nurses, and the present educational facilities which exist. Ann Wolf, R.N., Director of School of Nursing and Nursing Service, New York Hospital, New York City, gave a concise report of the activities of the Education Committee of the National League of Nursing Education in their efforts to increase and unify the standards which should be accepted as approved training for the profession of nursing. Miss Wolf traced the development of the Committee's work since 1917, calling special attention to the increased demands for a more general and thorough education of nurses.

Friday morning Miss Eleanor Jones, President of the Massachusetts State Association of Record Librarians, Newton Hospital, Newton, gave a short paper on the wishes which all record librarians in hospitals have respecting their work. It seems that the lack of coöperation of the medical staffs of the various hospitals and the failure of a standard classification of diagnoses cause most of their troubles.

Miss Ruth Tartakoff, Supervisor of the Admitting Office, New Haven Hospital, New Haven, Conn., and Miss Mary H. Roberts, Director of Social Service, Holyoke Hospital, Holyoke, Mass., discussed the need of trained Social Service workers in hospitals as aids to physicians in the care of patients.

Friday afternoon was devoted to a thorough discussion of Group Hospitalization and the prepayment plans for hospital services. Mr. Graham Davis, of the Duke Foundation, Charlotte, North Carolina, presented the state-wide development of prepayment plans in North Carolina in comparison with those in Great Britain. Mr. Frank Van Dyk, Executive Director of the Associated Hospital Service of New York, gave a clear discussion of the plan which has been in operation in New York for the past nine months. Of 57,000 subscribers in New York nearly 2,000 have had hospital service in nine months. These people have expressed their satisfaction with the plan. In the discussion which followed the presentation of these papers, definite statements were made that physicians in these communities are having better collections of accounts than before the hospitalization plans went into effect.

A most interesting development at the annual meeting was the establishment of a section for Trustees of Hospitals. Eighty-three trustees were in attendance and discussed their various problems in providing adequate hospital care for the patients.

During the meeting the Massachusetts members of the Association organized a State Association, the object of which is to promote the effectiveness of hospitals in Massachusetts.

Papers by Dr. Wilinsky and Dr. Faxon were read and are appended.

THE MASSACHUSETTS STATE HEALTH SURVEY*

BY CHARLES F. WILINSKY, M.D.,

Deputy Health Commissioner, City of Boston, Executive Director, Beth Israel Hospital

Late in November of 1934, shortly following the election of His Excellency, Governor Curley, a special committee was appointed by the Massachusetts Central Health Council to consider a study of the Public Health practices and a revision of the Public Health laws of Massachusetts. This committee had in mind a similar study conducted in the State of New York by a special Health Commission appointed in 1930 by President Roosevelt, who was at that time Governor of the Empire State. The conclusions and recommendations of the New York Commission proved of great value in raising the Public Health standards and practices in that state.

The hope was expressed at the meeting of the special committee of the Central Health Council that His Excellency the Governor might favor the legislative action necessary for the appointment of a Commission by the Governor for the study and revision of our Public Health laws. In view of the fact that the New York study was financed in the main by the Milbank Fund, it was suggested that an effort ought to be made to obtain necessary funds for the proposed Massachusetts study from a private foundation. Dr. Wilinsky agreed that, should there be a favorable reaction on the part of the Governor, he would approach the Commonwealth Fund of New York for the required funds.

His Excellency the Governor was most interested in this proposed study and embodied in his inaugural address this statement:

"The Commonwealth of Massachusetts has always maintained an enlightened interest in matters pertaining to the health of its citizens. It has been expedient from time to time to establish and maintain essential service, and to enact or amend laws for enlarging the scope of those departments created for the prevention of disease, since they affect the very lives of our people, and should be administered with the highest possible degree of efficiency. I have received assurances that in the event of the appointment of a commission for the study and revision of the public health laws of the Commonwealth, a national foundation interested in public health will defray the expenses of the commission, and, subject to favorable action by your Honorable Body, I shall appoint such a commission."

Dr. Wilinsky and Professor Wilson Smillie called on the Commonwealth Fund and secured from its representatives the assurance of their willingness to finance this project subject to the approval of the Board of Directors of the Fund. In April of 1935, the Board approved the grant of $10,000 for the financing of the proposed study.

The following resolve was introduced into the Legislature and passed:

> "Resolved, That an unpaid special commission, consisting of the commissioner of public health and the commissioner of mental diseases, ex officiis, and ten other members to be appointed by the governor, is hereby established for the purpose of studying and investigating the public health laws and policies of the commonwealth. After completing said study and investigation, but not later than the first Wednesday in December in the current year, said commission shall report to the general court by filing with the clerk of the House of Representatives, the results of its study and investigation, with its recommendations, if any, as to what changes it deems necessary in such laws and policies, together with drafts of such legislation as may be necessary to carry such recommendations into effect."

This resolve was passed in May, 1935, resulting in the appointment by His Excellency the Governor of the following Commission:

> Professor Curtis M. Hilliard—President, Massachusetts Health Council; Professor, Biology and Public Health, Simmons College.

*Presented at the meeting of the New England Hospital Association, February 27, 1936, Hotel Statler, Boston.

Professor Wilson G. Smillie—Professor, Public Health Administration, Harvard School of Public Health.

Dr. Alexander Begg—Dean, Boston University School of Medicine; Acting Secretary,* Massachusetts Medical Society.

Professor Samuel C. Prescott—Dean of Science and Professor of Industrial Biology, Massachusetts Institute of Technology.

Dr. Dwight O'Hara—Chairman, Public Health Committee, Massachusetts Medical Society; Professor of Preventive Medicine, Tufts College Medical School.

Dr. David Scannell—Chief of Surgical Service and President of Senior Staff, Boston City Hospital.

Dr. Francis X. Mahoney†—Health Commissioner, City of Boston.

Dr. Charles F. Wilinsky—Deputy Health Commissioner, City of Boston; Executive Director, Beth Israel Hospital.

Dr. Gerardo Balboni — Physician, Home for Italian Children; Member of staff of Massachusetts General Hospital.

Dr. Charles E. Mongan—President, Massachusetts Medical Society.

After its appointment, the Commission met and elected these officers:

Dr. Henry D. Chadwick, Chairman.
Dr. Wilson G. Smillie, Vice-Chairman.
Dr. Charles F. Wilinsky, Secretary-Treasurer.

The Commission set up fourteen committees to investigate major Public Health problems and procedures. Over one hundred men and women, leaders in their fields, are engaged in the study of the following topics:

1. Transference of vital statistics to the official health agency.
2. Formulation by the State Department of Public Health of statewide quarantine requirements.
3. Transference of municipal and county tuberculosis hospitals to the State.
4. Suitable standards for public health personnel.
5. The development of measures which may serve to take public health more nearly out of the field of politics.
6. Development of a more effective program for the control of gonorrhea and syphilis.
7. The development of adequate resources for rural communities, commensurate with their ability to pay.
8. Development of adult hygiene.
9. Better coördination of the school health services.
10. Formulation of reasonable laws for the suppression of rabies.
11. Formulation of a program for the reduction of maternal mortality.
12. Development of more effective health standards for industry.
13. The licensing of hospitals.

Of particular interest to hospitals are these subjects:

1. Licensing of hospitals.
2. Who is responsible for the payment of bills incurred in the care of the indigent in time of sickness in hospitals and in the home, also bills for the care of the injured in accidents, i.e., auto accidents.
3. Abuse of hospital charity, question of legislation making it a misdemeanor to obtain such under false pretense.
4. Responsibility of public health agencies for preventive medicine.
5. Statistics.
6. Miscellaneous—are there other subjects for consideration?

A number of the appointed committees have already submitted their reports to the Commission. Others are still working in subcommittees on various aspects of their particular studies.

There can be no question that the ultimate recommendations of the Commission will do much to strengthen Public Health work in Massachusetts and consequently to improve the health and well-being of the inhabitants of the state.

*In June 1935 elected as Secretary.
†Died January 14, 1936.

HOSPITAL COUNCILS*

BY NATHANIEL W. FAXON, M.D.,
Director, Massachusetts General Hospital and Massachusetts Eye and Ear Infirmary

Hospital Councils have developed from Superintendents' Councils or Clubs or other unofficial groups. The first Hospital Council, as such, that I know of was the Cleveland Hospital Council, established in 1915. The success of this Council and the very evident need of such organizations led to the formation of other Councils throughout the country. Some of these Councils were formally incorporated, included practically all the hospitals of the city as members, were intimately associated with health and welfare organizations and took an active part in community life. Others were small, informal gatherings of hospital superintendents who met to exchange information on matters of internal administration.

The American Hospital Association, believing that Hospital Councils were destined to play a part in the growth and development of hospitals from a community standpoint and in their relationship to Health and Welfare Departments, recommended, in 1932, that it be a subject for consideration by the Council on Community Relations and Administrative Practice (as it was then called). Since then the Council has made several reports upon the number, formation and accomplishments of Hospital

*Presented at the meeting of the New England Hospital Association, February 28, 1936, Hotel Statler, Boston.

Councils and has offered suggestions regarding the formation of Councils.

Although many and varied types of Hospital Councils have been found to exist, it can definitely be stated that their functions fall into two groups:

A. Coöperation among hospitals in dealing with common problems of internal administration.

B. Coördination of hospitals in action on community relations.

Under the first group, A, the following activities appear:

(a) Promotion of uniform statistics and accounting.

(b) Collection and dissemination of information, cost of supplies, wages.

(c) Collection and dissemination of information concerning rates.

(d) Coöperative purchasing.

(e) Coöperative action in collecting.

(f) Development of common standards of admission to wards and Out-Patient Departments.

Under the second group, B, there develops common action:

(a) In relation to legislation.

(b) In relation to publicity.

(c) In relation with other agencies, e.g., with the medical profession, local tax appropriating bodies, other governmental departments, industrial or insurance agencies.

(d) Common action in estabiishing Group Hospitalization.

(e) Relations with fund-raising organizations.

(f) Coördination with hospitals in other communities in the same region.

(g) Participation in community hospital and health surveys.

(h) Participation with other community groups in dealing with questions of the amount and distribution of hospitals and out-patient facilities, size and location of new hospitals and proposed additions.

These items, of course, do not cover all the fields of activity but are set down merely to indicate what Hospital Councils have already taken up advantageously.

I want to call your attention particularly to this last function because in the opinion of some, who have given thought to these matters, this will ultimately prove to be the most important activity of Hospital Councils. We are supposed to be living in a civilized community, and civilization in one sense consists in giving up voluntarily some of your rights in order that you may live peaceably with your neighbor, he of course doing likewise. Now what is applicable to individuals should also apply to institutions, for instance to hospitals. None of us as individuals, or as we represent institutions, are willing to sacrifice all our autonomy, nor is it needful, but we must recognize that there is a community welfare as clearly as there is a personal or institutional welfare. In every city there can be pointed out the mistakes in hospital building that have been made because the viewpoint and welfare of the community have been sacrificed to the ideas and plans of the individual organization or institution. From this formation of Hospital Councils the American Hospital Association has hoped to promote the consideration of hospital problems upon the broad basis of the welfare of the community.

The American Hospital Association Council also recommended that the term Hospital Council should be applied to an organization only when it included representatives of hospital departments other than the administration, as for instance, Trustees, Staff, and others, and also representatives of community interests. For organizations consisting only of Hospital Superintendents the term "Superintendents' Conference" was recommended. Where a number of small communities, covering a considerable area, had formed a joint organization having the same purposes as a Hospital Council, that it be called a "District Association." The latest report of the Committee, 1935, shows:

24 Local Hospital Councils.
13 Superintendents' Conferences.
7 Regional or District Associations.

These are all active organizations. Undoubtedly there are many more informal groups.

The Hospital Council of Boston was formed January 30, 1935, by action of twenty-three Boston hospitals. For many years there had been an informal Hospital Superintendents' Club but it was evident that a more formal organization was necessary—one which should include other community elements and which could represent the hospitals in relationship to the Health League, Council of Social Agencies, and the Community Federation.

"The object of the Hospital Council of Boston is to promote intelligent planning and coördination in the field of community hospital service; to serve as a forum for the discussion of common problems and as a clearing house for the exchange of information looking to the advancement of service; to interpret to the public functions of hospitals and their place in the community; to coöperate with other agencies concerned with health and social problems and such other business as may properly come before the Hospital Council."

Each member hospital was to have three representatives, a Trustee, the Superintendent, and—this was an unusual feature — a representative of the medical staff. Besides this representation from member hospitals there were also representatives from the Massachusetts Medical Society, the Dental Society, the Nurses' Association and five persons representing the public at large.

A close tie-up with the Boston Health League and the Council of Social Agencies was effected. The Executive Secretary of the Health League was made Executive Secretary of the Hospital Council,

giving half-time to each position. These three agencies occupy adjoining offices with many services and activities in common. This is economical and keeps the different organizations accurately informed of what the others are doing and so avoids wasteful duplication and loss of time.

It is hard to evaluate the Council as yet; it is too young. Perhaps if we look back over some of the items set down as proper functions for a Hospital Council and see how many we can check off, we may get some idea of the activities of the Boston Hospital Council during its first year.

Under those functions listed as coöperation among hospitals in dealing with common problems of internal administration, we find the following:

(a) Uniform accounting and reporting: undertaken to assist relations with the Community Fund and Federation.

(b) A start upon the subject of common rates and charges, through the appointment of a committee to coöperate with the Massachusetts Medical Society in adjusting Workmen's Compensation rates with the Insurance Companies and the Industrial Commission.

(c) Consideration by the Executive Committee of common standards for out-patient admissions, though it is only fair to state that nothing much has been accomplished yet.

Under those functions listed as coördination of hospitals in action on community relations, the following:

(a) Legislation: Just at present this item has occupied our attention almost to the exclusion of all others. Opposition to Bills requiring open staffs, Bills stipulating procedures in surgical operations, Bills taxing hospital property, and others have kept the Executive Committee busy. They have also sponsored the introduction of two Bills—one an amendment to the labor law requiring weekly payment of wages, which would exempt hospitals, and also a Bill sanctioning the incorporation of nonprofit hospital service corporations for the providing of hospital insurance to groups. In appearing before legislative committees it is a real help to be able to say that you are representing twenty-three hospitals which form the Boston Hospital Council. That represents votes and compels attention.

(d) Group hospital insurance: A committee is working on this; they have submitted a Bill, House 573. Ask your Representatives and Senators to support it. This applies to all hospitals in Massachusetts.

(e) Relations with the Community Federation are close and friendly. It is a help to both groups to have a central organization where many problems may be thrashed out in general terms before being taken to the individual unit for final consideration.

(g) Participation in surveys: These are with us always and the Council acts as a central clearing house in evaluating and collecting data. The one-day census taken under the direction of the Council was well done and the report, which will soon be published, will provide valuable factual data.

So you see we have made some progress. The adoption of uniform accounting and reporting of statistics to the Community Federation, the fostering of friendly relations with the Medical Society, the making of common endeavor with them on WCA problems, the beginning on Group Hospitalization Insurance, the opposing of dangerous legislation and the support of desirable bills have kept the Officers and Executive Committee very busy.

All in all, I think we can say that the Boston Hospital Council has helped; it has offered an organization through which Boston Hospitals may discuss, reach conclusions, and present their views in an effective manner. It forms a rallying and representative point. This is valuable and helpful. What the future will bring only time can tell.

BOSTON SOCIETY OF BIOLOGISTS

The February meeting of the Boston Society of Biologists was held at the Harvard Biological Laboratories in Cambridge, February 26, 1936. Dr. M. G. Banus spoke on The Final Tension in Isometric Muscular Contraction in Relation to Initial Tension and Length. By means of a special apparatus and careful measurements he was able successfully to separate the factors of tension and length in the isolated striated muscle. The final tension plotted against the initial stretch is a straight line function, but if the initial tension instead of the final tension is plotted, a curved line results. This was found to be true in several different striated muscles. Dr. Banus concluded that the final tension is a straight line function of the length of the fibre.

Dr. T. L. McMeekin spoke on Certain Solubility Relations of the Amino Acids and Their Derivatives. By neutralizing the different groups in the amino acids and studying the solubilities of the resulting compounds in water and alcohol, the effect of the neutralized groups was studied. The CH_2 group has a much smaller effect on solubility when it is between two polar groups than when it is a terminal group. The solubility in alcohol becomes greater when the chain of CH_2 groups attached to the carboxyl group becomes longer. When the NH_2 group is removed, the compound becomes less soluble in water and more soluble in alcohol. The effect is the same in all the mono-amino acids. These solubility tables are of great assistance in separating the amino acids and their derivatives.

Dr. H. L. Fevold spoke on the Augmentation of the Gonad Stimulating Action of Pituitary Extracts by Copper Salts. The follicle stimulating hormone of the pituitary stimulates the ovarian follicles to grow. The luteinizing hormone does not increase

the weight of the immature ovary when given alone, but when combined with the follicle stimulating hormone causes greater ovarian development than does the follicle stimulating hormone alone, and both follicles and corpora lutea develop. Thus there is a synergistic action of the two hormones.

Yeast extract and yeast ash increase the gonadotropic action of the follicle-stimulating hormone and also of follicle-stimulating hormone plus luteinizing hormone. Copper salts and zinc salts also produce increased responses but copper salts are more effective than those of zinc. Neither the yeast preparations nor the salts alter the qualitative ovarian response to the gonadotropic preparations.

Zinc salts probably bring about their effect by decreasing the rate of absorption of the active material but the effect produced by the copper salts cannot be explained on this basis. In normal immature rats, copper salts increase the action of both follicle-stimulating hormone and follicle-stimulating plus luteinizing hormone, but in hypophysectomized rats, they increase the action of follicle-stimulating hormone alone. Zinc salts produce the augmentation of both types of gonadotropic preparations in normal and in hypophysectomized rats. Copper salts also produce ovulation in rabbits while zinc salts do not. For these reasons it is assumed that the copper acts as a catalyzer between the follicle-stimulating hormone and the luteinizing hormone. Thus, if there is no hypophysis, there is presumably no luteinizing hormone in the blood with which the follicle-stimulating hormone can react. However, when the follicle-stimulating hormone and luteinizing hormone are injected together with copper into hypophysectomized rats, augmentation takes place as in the normal animals.

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held Monday, April 27, at 8:15 P.M., at the Boston Dispensary, 25 Bennet Street. Program: 1. The Significance of a Prominent S-Wave in Lead II of the Human Electrocardiogram. Dr. George Ravit. 2. Observations on Shock and Collapse. Dr. S. J. Thannhauser. 3. Unexplained High Basal Metabolism Rates in Heart Disease Without Failure. Dr. Heinz Magendantz. 4. Clinical Observations on the Treatment of Angina Pectoris. Dr. Joseph H. Pratt. 5. Some Effects of Diet Restriction on the Circulation in Patients with Heart Disease. Dr. Samuel H. Proger.

All members of the New England Heart Association and interested physicians are invited to attend.

JAMES M. FAULKNER, M.D., *Secretary.*

NEW ENGLAND SOCIETY OF PSYCHIATRY

The Annual Meeting of the New England Society of Psychiatry will be held at the Gardner State Hospital, East Gardner, Massachusetts, Wednesday, April 22, 1936.

The speaker will be Dr. A. Myerson, Professor of Neurology, Tufts College Medical School; Clinical Professor of Psychiatry, Harvard University Medical School, whose subject will be "The Neuroses."

HARLAN L. PAINE, M.D., *Secretary-Treasurer.*

THE AMERICAN ASSOCIATION FOR THE STUDY AND CONTROL OF RHEUMATIC DISEASES

The American Association for the Study and Control of Rheumatic Diseases will hold its fifth conference on rheumatic diseases at the Phillips Hotel, third floor, on May 11, at 9 o'clock, in Kansas City.

The tentative program appears below:

An Educational Program of the Differential Diagnosis of Diseases of Joints

1. Clinical Grouping and Diagnostic Approach to the Patient with Joint Conditions. Dr. Russell Haden.
2. Differential Diagnosis of Joint Diseases from the Standpoint of Pathology. Dr. Edwin P. Jordan.
3. The Essential Features in Differential Diagnosis of Atrophic and Hypertrophic Arthritis. Dr. Ralph Boots.
4. Differential Diagnosis between Strumpell-Marie Disease and Osteoarthritis of the Spine. Dr. Joseph L. Miller.
5. Differential Diagnostic Points of Gonorrheal Arthritis. Dr. Stafford Warren.
6. Differential Diagnostic Points of Tubercular Arthritis, Especially Tubercular Polyarthritis. Dr. Frank D. Dickson.
7. Differential Diagnostic Points of Rheumatic Fever. Dr. Ralph A. Kinsella.
8. Differential Diagnostic Facts about Gout, Distinguishing It from Other Joint Diseases. Dr. Philip S. Hench.
9. Differential Diagnosis of Traumatic Arthritis. Dr. Willis Campbell.
10. Differential Diagnosis of Fibrositis. Dr. C. H. Slocumb.
11. Differential Diagnostic Points of Constitutional Conditions, Mistaken for Arthritis, Which Produce Skeletal Aches and Pains. Dr. William J. Kerr.

THE AMERICAN NEISSERIAN MEDICAL SOCIETY

The American Neisserian Medical Society will hold its second annual meeting on May 18, 1936, in the Hotel Statler, Boston, Massachusetts. All who are interested are cordially invited.

PROGRAM

10:00 A.M. The "Flow of the Seed" in Antiquity. M. L. Brodny, M.D., Boston.

The Technique of Isolating the Gonococcus and of Determining the Thermal Death Time. C. M. Carpenter, M.D., Rochester.

The Application of the Thermal Death Time

Principle to the Treatment of Gonococcal Infection by Fever Therapy. S. L. Warren, M.D., Rochester.

The papers of Drs. Carpenter and Warren will be illustrated by moving pictures in colors.

Business meeting.

Report of Secretary-Treasurer.

Report of Auditing Committee.

Report of Nominating Committee.

Election of Officers.

Report of Executive Committee.

Vote on proposed amendment to the Constitution. Article III (Membership) Section I, Paragraph A, which reads: "Are residents of the United States or its Territories, Canada, or Mexico." To be amended to read: "Are residents of the United States or its Territories, Canada, Mexico, or islands adjacent thereto."

Election of members.

2:00 P.M. Presidential Address.

Hans Zinsser, M.D. (guest speaker). To What Extent Can a Bacteriologist Contribute to the Control of Venereal Diseases?

Reports of the following committees:

Male Clinical, P. S. Pelouze, M.D., Chairman.

Female Clinical, C. C. Norris, M.D., Chairman.

Laboratory and Research, C. M. Carpenter, M.D., Chairman.

Public Health, Thomas Parran, Jr., M.D., Chairman.

Sociological, W. F. Snow, M.D., Chairman.

Discussion and action on the following questions:

(a) Methods of obtaining funds for encouragement of research on the gonococcus and gonococcal infections.

(b) Publication of transactions.

(c) Work programs for the ensuing year.

7:30 P.M. Dinner: The Neisserian Medical Society of Massachusetts will be host.

MASSACHUSETTS SOCIETY FOR SOCIAL HYGIENE

Annual Meeting

The Annual Meeting of the Massachusetts Society for Social Hygiene will be held at the Hotel Vendome, Empire Room, Boston, on April 30.

Luncheon 12:30 P.M. $1.00.

Speaker: Rev. John Howland Lathrop.

Subject: Counselling Youth in Preparation for Marriage.

MASSACHUSETTS SAFETY CONFERENCE

The Fifteenth Annual Convocation of the Massachusetts Safety Conference, of which Manfred Bowditch, Director of the Massachusetts Division of Occupational Hygiene, is chairman, will be held at the Hotel Statler, Boston, April 23-24, 1936. The meeting will begin at 2.00 P.M. and will include a section on Industrial Health Problems.

"Radium Poisoning" Demonstration. Robley D. Evans, Ph.D., Assistant Professor of Physics, Massachusetts Institute of Technology.

"Dust Control in the Small Granite Plant." Edward C. Riley, Supervising Engineer, Massachusetts Granite Dust Control Project.

"The Nose as a Dust Filter." Philip Drinker, Associate Professor of Industrial Hygiene, Harvard School of Public Health.

Discussion, led by Anthony J. Lanza, M.D., Assistant Medical Director, Metropolitan Life Insurance Company.

The Granite Dust Control Project being conducted by the Division in coöperation with the Massachusetts Works Progress Administration is now nearing completion. As a demonstration of practical dust control, it is of interest to everyone concerned with the industrial dust problem.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance), Tuesday evening, April 28, at 8:15 P.M.

Program

Presentation of Cases.

The Pathology of Vitamin Deficiencies. By Dr. S. Burt Wolbach.

Medical students and physicians are cordially invited to attend.

Marshall N. Fulton, M.D., *Secretary.*

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

The 310th meeting of the New England Ophthalmological Society will be held on Tuesday, April 21, 1936, at the Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston.

9:00 A.M. Clinic and Operating Room.

11:30 A.M. Neuro-Ophthalmological Conference.

8:00 P.M.

Cases:

Dacryo-Adenitis—Dr. Benjamin Sachs.

Tumors of Lids and Conjunctiva—Dr. Benjamin Sachs.

Ocular Paralysis Following Spinal Anesthesia—Dr. James J. Regan.

Monocular Pigmentary Degeneration of Retina—Dr. James J. Regan.

Address:

The Changes that Occur in a Detached Retina—Dr. Algernon B. Reese (New York City).

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, APRIL 20, 1936

Tuesday, April 21—

New England Ophthalmological Society. Massachusetts Eye and Ear Infirmary, Boston.

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. X-Ray Demonstration. Dr. Alice Ettinger.

*12 M. South End Medical Club, office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

2:30 P.M. Pediatric Ward Visit. Massachusetts Eye and Ear Infirmary.

8:15 P.M. Boston Medical History Club. Boston Medical Library, 8 Fenway, Boston.

Wednesday, April 22—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Pericarditis. Prof. G. Klemperer.

†12 M. Clinico-Pathological Conference. Children's Hospital.

4 P.M. The George Washington Gay Lecture. Amphitheatre at Tufts College Medical School.

Thursday, April 23—

*8:30-9:30 A.M. Clinic, Surgical Staff of the Peter Bent Brigham Hospital, at the Peter Bent Brigham Hospital.

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Social Service Case Presentation. Miss E. R. Canterbury.

Massachusetts Safety Conference. Hotel Statler, Boston.

*3:30 P.M. Medical Clinic at the Peter Bent Brigham Hospital.

Friday, April 24—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Autonomic Pharmacological Experiments on the Human Being. Dr. Abraham Myerson.

Massachusetts Safety Conference, Hotel Statler, Boston.

12 M. Massachusetts General Hospital. Clinical meeting of Staff of the Children's Medical Service. Ether Dome.

Saturday, April 25—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

*10-12. Staff rounds at the Peter Bent Brigham Hospital.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

April 17—Boston University School of Medicine Surgical Clinic at the Boston City Hospital. 12-1, Cheever Amphitheatre.

April 20-24—A Postgraduate Institute in Philadelphia. See page 497, issue of March 5.

April 21—New England Ophthalmological Society. See page 812.

April 21—Boston Medical History Club. Boston Medical Library, 8 Fenway, Boston, at 8:15 P.M.

April 21—South End Medical Club. Office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, at 12 M.

April 22—The George Washington Gay Lecture. See page 803.

April 22—New England Society of Psychiatry. See page 811.

April 23—Medical Clinic, Peter Bent Brigham Hospital. See page 803.

April 23—Clover Hill Hospital. See page 803.

April 23-24—Massachusetts Safety Conference. See page 812.

April 27—New England Heart Association. See page 811.

April 27—Springfield Medical Association, 8:30 P.M. at the rooms of the Springfield Academy of Medicine, 20 Maple Street. The Development of Non-Surgical Specialties. Dr. Allen S. Johnson. The Development of Surgical Specialties. Dr. Eugene W. Beauchamp.

April 28—Harvard Medical Society. See page 812.

April 29—Annual Tufts Alumni Address. See page 803.

April 29 - May 9—Physicians' Art Exhibition. See page 803.

April 30—Massachusetts Society for Social Hygiene. See page 812.

April 30 - May 1—The Henry Jackson Lectures Offered by the New England Heart Association. See page 803.

May 1, 2, 3, and 4—The American Association on Mental Deficiency. See page 610, issue of March 19.

May 11—American Medical Golfers Play in Kansas City. See page 710, issue of April 2.

May 11—American Association for the Study and Control of Rheumatic Diseases. See page 811.

May 12—American Heart Association, Inc. See page 712, issue of April 2.

May 12-16—The International Congress of Physical Medicine. See page 443, issue of February 27.

May 18—The American Neisserian Medical Society. See page 811.

May 31 - June 1—International Cardiological Meeting, Royat (Auvergne) Assembly of Physiologists, Pathologists and Therapeutists. See page 754, issue of April 9.

June 15-19—The Executive Board of the Catholic Hospital Association will meet at the Fifth Regiment Armory, Baltimore, Md.

June 16-July 28—Summer Course in Bacteriology. See page 385, issue of February 20.

September, 1936—First International Conference on Fever Therapy. See page 1325, issue of December 26, 1935.

September, 1936—First International Congress of Sanatoria and Private Nursing Homes. See page 803.

September 7-10—International Union against Tuberculosis. See page 554, issue of March 12.

October 19-23—Clinical Congress of the American College of Surgeons. See page 180, issue of January 23.

DISTRICT MEDICAL SOCIETIES

BRISTOL NORTH DISTRICT MEDICAL SOCIETY

April 16—Annual Meeting. Hotel Taunton, 6 P.M.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

May 7—Thursday. Censors' Meeting.

May 13—Wednesday. Annual Meeting. Salem Country Club. Dinner at 7 P.M. Speaker: Dr. Paul White. Subject to be announced later.

R. E. STONE, M.D., Secretary.
88 Lothrop Boulevard, Beverly.

FRANKLIN DISTRICT MEDICAL SOCIETY

May 12—Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

May 6—Bear Hill Golf Club, Stoneham, at 12:15 P.M.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

May—Annual Meeting. (Place, date and subject to be announced.)

The censors meet for the examination of candidates: May 7, 1936; November 5, 1936.

FRANK S. CRUICKSHANK, M.D., Secretary.
1236 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

April 16—Brockton Hospital.
May 21—Lakeville State Sanatorium.

G. A. MOORE, M.D., Secretary.
167 Newbury Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

April 29—Boston Medical Library, 8 Fenway, Boston. 8:15 P.M.

ROBERT L. DeNORMANDIE, M.D., President,
CHARLES C. LUND, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

May 7—Censors' Meeting. See page 713, issue of April 2.

May 13—Wednesday afternoon and evening. Annual Meeting of Society. Time, place and details of program to be announced.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

April 22—Burbank Hospital, Fitchburg.

BOOK REVIEW

Localized Rarefying Conditions of Bone as Exemplified by Legg-Perthes' Disease, Osgood-Schlatter's Disease, Kümmell's Disease and Related Conditions. E. S. J. King. 400 pp. Baltimore: William Wood & Company. $7.50.

Within the 400 pages of this well-illustrated book the reader will find an able discussion of those related lesions to which, unfortunately, the names of their original reporters have become attached. This purely historic nomenclature connotes different disease entities, whereas the evidence strongly suggests that most of these "localized rarefying conditions of bone" represent only different anatomic localizations of a single syndrome. Much of the material is taken from the author's Royal College of Surgeons' Jacksonian prize essay of 1933.

Section I deals with "The Nature of Bone." King believes that the conception of metaplasia of various connective tissues is fundamental in the appreciation of the changes in bone. Two theories are possible: (1) that bone is laid down and controlled by cells and (2) that bone exists or is laid down irrespective of cells. The conclusion is reached that osteoblasts, and of course bone cells, are a fundamental feature of bone structure. The nature and function of the intercellular substance still remain somewhat obscure, but it seems to "live" even if it is not "living" in the sense that cells are living. The exact chemical composition of bone still needs study, but the hypothesis of Gebhardt still holds that the calcium of "areas" of calcification is in a crystalline state while the calcium of bone is in a colloidal phase. In spite of previous views, there is evidence to indicate that often a real ossification in or of the cartilage occurs. Bone is one of the most labile and not one of the most fixed tissues of the body. "The nature and causes of calcification are still shrouded in mystery."

The nature of rarefaction of bone is discussed. Differing from the views of Leriche and Policard, King holds that the relationship of the vascular changes to the resorption is not that of cause and effect but a physiological relationship.

Pathological calcification and heterotopic ossification are also discussed and the recently advanced hypothesis that the enzyme phosphatase is responsible for the formation of bone is supported. Some of the generalized diseases of bone are then considered and the relationship of roentgenology to bone changes. Anatomical considerations conclude Section I.

In Section II, under the inclusive name of "Osteochondritis Juvenalis", are reviewed the essential features of the syndrome in the different anatomic areas in which it has been noted. The condition may be present only when developing bone is in the transition stage from cartilage to bone and ceases when ossification is complete. It does not start until cartilage begins to ossify. Etiologically, King believes that the condition may be incidental to a number of different causes rather than to a single specific cause. If the author possesses a judicial mind (and in the opinion of your reviewer there is strong evidence that he does), this conclusion is worthy of attention since the review of the literature seems to have been most thorough. In the bibliography of osteochondritis of the upper epiphysis of the femur, for example, references are given to four hundred and twenty-six publications. Following the thirty pages concerning this, perhaps the most clinically important manifestation of osteochondritis juvenalis, consideration is given to the other common locations of this condition. In suggestions as to treatment which follow the headings of pathology, diagnosis and etiology, there is perhaps too much emphasis placed on the necessity for complete immobilization for long periods. Most recent evidence (e.g., Danforth, M. S., "Treatment of Legg-Calve'-Perthes' Disease Without Weight-Bearing", Journal of Bone and Joint Surgery, **16**: 516-534 July '34) suggests that long freedom from weight-bearing, at least, may be justified in this particular manifestation because most complete anatomic and functional recovery seems to be favored thereby. In this region this is of extreme importance if the chronic arthritic changes of later life are to be prevented. However, any unnecessarily prolonged interference with function is most undesirable. The extreme muscular atrophy which ensues may never be entirely overcome. Where the condition does not involve joint mechanisms, e.g., in the lesions of the adolescent tibial tubercle and the earlier "apophysitis" of the os calcis, the reviewer's experience leads him to believe that complete prohibition of function is very rarely necessary. The symptoms may be entirely relieved by intelligent adhesive plaster strapping and a completely satisfactory end-result obtained while the patient remains ambulatory.

Section III deals with "Post-Traumatic Rarefaction" of bone in both its diffuse and circumscribed forms. The most tenable etiological theory in the diffuse form the author considers to be "some change in the local nervous system, possibly acting by way of the blood vessels". Kümmell's disease is the most familiar form of this type. The circumscribed type is exemplified by such manifestations as "Kienböck's disease" of the carpal semilunar.

Section IV discusses "Rarefaction of Articular Surfaces Associated with Loose Bodies." The most commonly recognized lesion of this nature is "osteochondritis dissecans" of the knee joint. No completely satisfactory etiological explanation is advanced, nor are any routine methods of treatment advocated. Conservatism in the early stages and in the absence of disabling symptoms is favored.

The book is well worth careful reading. The author's style is clear. He avoids both the hazard of dogmatism and the pitfall of indeterminism. Whenever he considers a personal opinion justifiable, it is fearlessly advanced. We highly commend the book to both internists and surgeons. Every orthopedic surgeon should familiarize himself with its contents.